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CONTRIBUTIONS

TO

CLINICAL AND PRACTICAL

MEDICINE.

BY

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CONTRIBUTIONS TO MEDICINE.

CHAPTER I.

RESEARCHES ON THE TEMPERATURE IN PHTHISIS AND SOME OTHER DISEASES.*

GENTLEMEN,

Of the many symptoms of disease, there is no one more interesting or more worthy of attention than the temperature of the body. The wonderful power which the human economy possesses of maintaining in health an uniform, or almost uniform temperature under varying conditions of external heat, is a phenomenon interesting alike to the student of natural history, the chemist, the physiologist, and the physician; and it is nowise remarkable that the variations of temperature produced by abnormal conditions should have attracted the notice of early observers. As far back as 1638, Sanctorius applied the thermometer to the determination of temperature; and a century later Van Swieten, by improved instruments, made observations which proved that in fever the temperature was higher than in health. De Haen, a colleague of Van Swieten, made further investigations, and became aware of the morning remissions and even-

* An Address (with additions) delivered to the Members of the Liverpool Medical Institution.

ing exacerbations which characterise fevers; of the rise of temperature in febrile rigors; and of the discrepancies between the pulse and the temperature. He used his knowledge of these changes as a guide to his therapeutics and regarded the return to a normal temperature as a proof of convalescence. No observations of any great value to practical medicine seem to have been made after De Haen's time, until Currie made his important researches; but, although Currie's work on this subject went through three editions, it influenced his contemporaries very slightly. From the time of Currie to the year 1840, but little may be said to have been done in medical thermometry.

At that time, there was commenced a series of painstaking investigations which have never been interrupted. It was not, however, till 1851 that Wunderlich began his researches, which resulted in the treatise so well known to the profession; and thus it appears that, with a few exceptions, to some of which I have referred, it was reserved for the practitioners of the latter half of the nineteenth century to bring to bear instruments of precision in the investigation of this particular phenomenon of disease, and to study it scientifically.

When I began the study of the profession no regular temperature observations were made in our hospital wards, and the clinical thermometer was not the constant accompaniment of the practitioner in his rounds. Of late years, however, the instrument has become one of daily use, and from an

accurate record of temperature observations in disease, attempts have been made to generalise on this symptom.

That in the early days of the introduction into practice of a novelty, its value should be either overestimated on the one hand, or somewhat doubted on the other, is only what might be expected. Possibly the use of the thermometer by the inexperienced may have been to some extent productive of error. The recognition of the fact that the temperature had risen a few degrees above the normal may have led to an exaggeration of the patient's danger, and the fear that some serious pyrexial condition was present; whilst, on the other hand, a fall of temperature below the normal may have given rise to the notion that collapse was threatened. The prolonged use of the thermometer has, however, enabled us to avoid errors of this kind—errors which, as they were on the side of caution, were of a very venial kind.

The facts to which I wish to call your attention are the outcome of a long series of careful observations, made either by myself, or under my supervision, and they may, I believe, be implicitly relied on. I have thought, therefore, that they might be worthy of being made the subject of a discourse which it is my duty, as your president, to address to you on this occasion.

Although the normal temperature of the human body is placed at 98·4 to 98·6 deg. Fahr., this is liable to certain variations, which should be borne

in mind in thermometric observations in disease. With regard to adults, it is stated by Davy, Gierse, Fröhlich, and Lichtenfels, as the result of observations on themselves, that there is a daily range of about one degree. Sydney Ringer, as the average of a series of observations, makes the range about the same. My own observations on myself give 98·6 deg. as the highest reading, and 97 deg. as the lowest, thus giving a daily range of 1·6 deg.

In children, the daily range is greater; and it is a noticeable fact, that the range diminishes with increasing years; so that in the aged the range is very slight. There appears to be in all persons, of whatever age, a tendency to a fall of temperature in the evening; and in children this amounts to one, two, or three degrees, and begins usually from 5 to 7 p.m., and is rapid up to 9 or 10 p.m. The minimum is reached during the early hours of the morning, from 2 to 4 a.m.; and then a rise begins, which may reach its maximum at 9 or 10 a.m., but sometimes later. It is very important to bear in mind these alterations of temperature in the diagnosis and treatment of the diseases of children; for, as there is a normal rise at certain periods of the day, a single examination at such times might possibly lead to error; and, again, inasmuch as there is a normal fall late in the evening, the recognition of a persistent high temperature at that time might aid us very much in the diagnosis of certain cases of tubercular and enteric fevers, often very difficult diseases to make out in children.

Another question with reference to normal temperature is, whether there is any difference in the temperature in different climates. Dr. John Davy, from observations made on himself, stated that in tropical climates the normal temperature of man was one degree higher than in temperate zones. This statement remained for a long time unchallenged; but of late it has been apparently confuted by the observations of two army surgeons, Drs. Johnson and Boileau. Without referring in detail to their observations, it may suffice to say they have found that the temperature of the human body does not rise under the influence of tropical heat. Dr. Johnson states that the mean of 320 readings was 97·74 deg. Fahr.; and it would thus appear that the normal temperature is rather lower in intertropical, than in temperate climates. At all events, we may, I think, safely conclude that it is not higher, and therefore we must be prepared to recognise a temperature of 99 to 100 deg. as abnormal in both climates.

The first disease to which I wish to call your attention in connection with this subject is phthisis. The development and progress of this affection are marked, as a rule, by elevations of temperature, sometimes of a very striking character. How far these are due to *tuberculosis* or *tuberculisation*, to the phthisical diathesis, or to inflammatory or other changes incident thereto, or consequent thereon, I cannot at the present moment stop to consider. There are, I believe, no diseases, except ague, and

septicæmia, including pyæmia, in which there are alterations of temperature so extreme, characterising the pyrexial and apyrexial states, or the periods of exacerbation and remission, as in those which come under the designation of acute phthisis; and, as far as my own experience goes, it is in the pneumonic form of the disease that the most marked oscillations of temperature are observed.

Table No. I shows the temperature of a lady whom I attended. The case was one in which there was a rapid consolidation of the apices of both lungs, with all the characteristics of a scrofulous pneumonia, a rapid formation of cavities, and death. The temperature was taken morning and evening; the highest temperature recorded was taken at 9 p.m. of the 17th June, when it was found to be 106·6 deg. Fahr. On the following morning, at 10 o'clock, it was 97 deg., giving a fall of 9·6 deg. The lowest temperature observed was 96 deg. on several mornings. This extreme oscillation—a very high evening temperature, and a low or subnormal morning temperature—is a marked characteristic of acute phthisis, especially of the pneumonic form. It is very rare, however, to find the oscillations as great as in the case now under consideration.

TABLE I.—*M. S., aged* 30. *Acute Pneumonic Phthisis.*

Date.	Morning.	Evening.
June 1, 1875	98·8	105·0
,, 2 ,, ...	97·4	104·2
,, 3 ,, ...	98·6	104·4
,, 4 ,, ...	99 0	101·5
,, 5 ,, ...	99·0	103·5

TABLE I.—*M. S., aged* 30. *Acute Pneumonic Phthisis*—continued.

	Morning.	Evening.
June 6, 1875	96·0	102·0
,, 7 ,, ...	97·0	100·0
,, 8 ,, ...	97·0	103·0
,, 9 ,, ...	96·0	101·8
,, 10 ,, ...	97·4	102·0
,, 11 ,, ...	96·0	103·8
,, 12 ,, ...	97·0	102·2
,, 13 ,, ...	96·5	102·2
,, 14 ,, ...	97·0	104·0
,, 15 ,, ...	97·0	104·2
,, 16 ,, ...	98·4	103·8
,, 17 ,, ...	98·4	106·6
,, 18 ,, ...	97·0	103·5
,, 19 ,, ...	99·1	103·0
,, 20 ,, ...	99·7	102·8
,, 21 ,, ...	96·0	103·0

On looking at a table of this kind, it is easy to understand how grave must be the nature of this disease; how the existence of so high a temperature, even for a few hours daily, must tend to a destructive waste of tissue. It is only in consequence of the fact that the temperature is not continuous, that there are remissions of the fever—amounting, indeed, to intermissions (for the temperature frequently falls below the normal)—that we can account for the maintenance of life.

Now let me call your attention to another case very similar to the last, but not marked by symptoms so acute. The patient whose temperature is represented in Table No. II was a woman twenty-eight years of age, who was admitted into the Infirmary under my care on December 28th, 1877. At that time, there was crepitation over the upper part of the left lung, and a temperature, three days after admission, of 103·6 deg. In this patient, there was rapid consolidation, and development of

cavities, in the upper part of both lungs. The woman died on February 4th, 1878. You will observe that the case was characterised by oscillations of temperature, which, although not as great as those of the preceding case, were nevertheless well marked. From the evening of the 16th of January, at 5 p.m. to 9 the following morning, there was a fall of five degrees and a half, from 105 to 99·5 deg., and again from 5 p.m. of the 24th to 9 a.m. of the 25th there was a fall of four degrees, from 105 to 101 degrees.

TABLE II.—*S. C., aged* 28. *Acute Pneumonic Phthisis.*

Date.	9 a.m.	5 p.m.	9 p.m.
January 16, 1878	103·0	105·0	104·6
,, 17 ,, ...	99·5	100·8	102·0
,, 18 ,, ...	102·6	103·6	103·6
,, 19 ,, ...	102·4	105·0	104·7
,, 20 ,, ...	103·4	104·7	103·8
,, 21 ,, ...	102·3	104·7	104·0
,, 22 ,, ...	102·4	105·0	104·6
,, 23 ,, ...	104·0	104·8	104·0
,, 24 ,, ...	103·0	105·0	102·5
,, 25 ,, ...	101·0	103·5	104·7
,, 26 ,, ...	103·0	104·6	104·0
,, 27 ,, ...	102·0	103·4	103·0
,, 28 ,, ...	100·5	102·6	102·6
,, 29 ,, ...	100·0	103·0	104·0

As a contrast to these two cases, let me call your attention to the Tables Nos. III and IV. These represent the temperature oscillations of two cases of acute miliary tuberculosis of the lungs, *acute tubercular phthisis.*

The first case (No. III) is that of a man who was admitted into the Infirmary, under my care, on the 6th of February, 1878, and died on the 14th. He was a fireman on board a steamer, and eleven

days before his admission into hospital he was seized with dyspnœa and faintness, and gave up work. On examination, I found the physical signs of cavities, which I thought were old, under the right clavicle, with subcrepitant *râles* all over both lungs. There was a somewhat congested state of the face, and a general condition not unlike that met with in typhoid fever. I considered the case to be one of acute tuberculosis with old-standing lung disease. This diagnosis was verified by the *post-mortem* examination. Some old cavities were found in the right apex; and both lungs were studded throughout their entire extent with semitransparent tubercles, with the usual engorgement of the pulmonary tissue around them.

As you will see, the temperature in this instance was not *very* high. It reached 105·6 deg. at five o'clock one evening; but a temperature considerably above the normal was persistently maintained. On no occasion—and it was taken three times a day—did it approach the normal; the lowest marked being 101·4 deg. The oscillations were far less marked than in the previous cases.

TABLE III.—*J. K., aged* 28. *Tubercular Phthisis.*

Date.	9 a.m.	5 p.m.	9 p.m.
February 6, 1878... ...	—	—	104·0
„ 7 „	103·6	103·8	103·0
„ 8 „	102·4	105·5	104·0
„ 9 „	103·2	104·1	104·0
„ 10 „	102·0	101·4	101·9
„ 11 „	103·0	103·2	103·5
„ 12 „	102·4	102·8	103·0
„ 13 „	103·0	105·0	103·0
„ 14 „	104·0	104·8	—

The second case (No. IV) was that of a man who was admitted into the Infirmary, under my care, on April 5th, 1873, and died on May 16th. At the necropsy, the lungs were found studded throughout with miliary tubercles, and some small cavities existed in the lower lobe of the right lung. My diagnosis of the case was "acute tubercular phthisis." Here, again, the temperature did not rise to an extreme height, nor were the daily oscillations very decided; the greatest fall from exacerbation to remission being 3·6 deg.

TABLE IV.—*W. H., aged* 24. *Acute Tubercular Phthisis.*

Date	Morning.	Evening.
April 20, 1873	102·2	104·2
,, 21 ,, ...	103·4	102·2
,, 22 ,, ...	102·8	103·3
,, 23 ,, ...	102·8	103·5
,, 24 ,, ...	102·8	103·0
,, 25 ,, ...	101·5	101·0
,, 26 ,, ...	102·2	103·5
,, 27 ,, ...	103·6	104·5
,, 28 ,, ...	103·2	103·8
,, 29 ,, ...	103·7	103·3
,, 30 ,, ...	102·0	102·5
May 1 ,, ...	101·5	103·2
,, 2 ,, ...	102·8	103·0
,, 3 ,, ...	101·5	102·5
,, 4 ,, ...	100·0	103·0
,, 5 ,, ...	102·4	103·2
,, 6 ,, ...	100·0	103·0
,, 7 ,, ...	99·4	103·0
,, 8 ,, ...	100·5	103·0
,, 9 ,, ...	102·2	103·6
,, 10 ,, ...	101·6	103·8
,, 11 ,, ...	102·5	103·8
,, 12 ,, ...	101·8	103·3

In the first two of these four cases of acute phthisis, there was a rapid destruction of tissue

going on, extensive softening and excavation of deposits thrown out into the lungs, and extreme exacerbations and remissions of temperature occurred. In the two latter, the destruction of tissue was little or none, but there was extensive deposit of tubercle — *tuberculisation ;* and this, although it raised the temperature considerably, and maintained it at a high level, was not attended by very great defervescence.

It is interesting to note these phenomena, but not easy to account for them. The cases of acute pneumonic phthisis, in their daily alternations of temperature, come to resemble very closely cases of septicæmia and pyæmia ; but here, again, there is a difference. In pyæmia, the exacerbations and remissions may occur three or four times a day ; whereas in acute phthisis there is, as a rule, a steady rise of temperature from the morning to the evening—the maximum being reached at five, six, seven, or nine o'clock—and then there is a steady fall. Table No. V shows the oscillations of temperature in a well-marked case of pyæmia, in which after death, numerous abscesses (pyæmic) were found in the liver. The elevations of temperature in pyæmia are attended with rigors ; and in this respect the disease resembles an intermittent fever. But in pyæmia, as is not the case in ague, there may be several elevations and falls of temperature—exacerbations and remissions—in the course of the day. The Table represents the variations of temperature during four days. The patient, a woman, had suffered from abscess

behind the ear, which had been opened. Hæmorrhage had occurred, for which the occipital artery was tied. She came under my care on the 22nd of March, 1878. At 11.30 a.m. on that day, the temperature was 106 deg.; at 9 p.m., 101·4 deg. The following day, at 9 a.m., it was 101·8 deg.; and at 9 p.m., 98·2 deg. The next day the temperature was taken frequently, and you will observe that there were two distinct exacerbations followed by remissions, amounting, indeed, to a cessation of fever—intermission.

TABLE V.—*M. C. Pyæmia.*

March 22, 1878	11.30 a.m.	...	106·0
,, ,, ,,	9 p.m.	...	101·4
,, 23 ,,	9 a.m.	...	101·8
,, ,, ,,	9 p.m.	...	98·2
,, 24 ,,	9 a.m.	...	102·5
,, ,, ,,	11.30 ,,	...	104·2
,, ,, ,,	4.30 p.m.	...	100·5
,, ,, ,,	6 ,,	...	98·5
,, ,, ,,	12 ,,	...	104·0
,, 25 ,,	9 a.m.	...	99·5
,, ,, ,,	5 p.m.	...	101·5
,, ,, ,,	9 ,,	...	101·5
,, 26 ,,	9 a.m.	...	100·0
,, ,, ,,	12 noon	...	101·0
,, ,, ,,	3 p.m.	...	102·5
,, ,, ,,	6 ,,	...	103·0
,, ,, ,,	9 ,,	...	101·5
,, 27 ,,	9 a.m.	...	101·0

Table No. VI shows the temperature of a case of septicæmia after excision of the breast, performed on November 13th. On November 19th, six days after the operation, the temperature rose to 105 deg. During the following day, nineteen observations were made. There were two rigors, and two distinct exacerbations of temperature, followed by

remissions. You will observe that there is a rise of temperature reported of 4·4 deg. in ten minutes—viz., from 102·6 deg. to 107 deg. I am told that this great rise was carefully noted at the time, and that such rises are occasionally met with in septicæmia. The patient died on November 22nd. No deposits of pus were found. I am indebted to Mr. Damer Harrisson for this chart.

TABLE VI.— ——, *aged* 48. *Septicæmia after Excision of the Breast.*

November 13	Evening	99·8
,, 14	Morning	99·5
,, ,, ...	Evening	100·0
,, 15	Morning	99·4
,, ,, ...	Evening	99·7
,, 16	Morning	99·8
,, ,, ...	Evening	100·9
,, 17	Morning	101·6
,, ,, ...	Evening	101·0
,, 18	Morning	100·5
,, ,, ...	Evening	101·5
,, 19	Morning	102·8
,, ,, ...	Evening	105·0
,, 20	8 a.m.	103·6
,, ,, ...	11 a.m.	104·0
,, ,, ...	12.15 p.m.	105·0
	(12.30 p.m., rigor)	
,, ,, ...	1 ,,	106·2
,, ,, ...	1.30 ,,	105·7
,, ,, ...	1.45 ,,	104·5
,, ,, ...	2 ,,	104·0
,, ,, ...	2.45 ,,	103·0
,, ,, ...	3 ,,	102·8
,, ,, ...	4 ,,	102·6
,, ,, ...	4.30 ,, (rigor)	102·6
,, ,, ...	4.40 ,,	107·0
,, ,, ...	5.15 ,,	105·2
,, ,, ...	5.30 ,,	104·5
,, ,, ...	6 ,,	104·0
,, ,, ...	6.30 ,,	103·5
,, ,, ...	7 ,,	103·4
,, ,, ...	7.30 ,,	102·5
,, ,, ...	8.50 ,,	100·5
,, 21	Morning	100·4
,, ,, ...	Evening	105·0
,, 22	Morning	103·0

Table No. VII shows the temperature-oscillations in a case of septicæmia connected with phlebitis. The patient was a woman who was admitted under my care into the Infirmary, and was discharged, well, after several weeks' residence in the institution. Her symptoms, except as regards the rises of temperature, were not acute. Her pulse, except at first, was not very quick, and the elevations of temperature were not attended with marked rigors.

TABLE VII.—*M. J. Phlebitis: Septicæmia.*

November	21,	1878	...	...	5 p.m.	103·0
,,	,,	,,	...	...	9 ,,	101·0
,,	22	,,	...	...	9 a.m.	97·2
,,	,,	,,	...	...	1 p.m.	98·7
,,	,,	,,	...	...	1.40 p.m	101·2
,,	,,	,,	...	...	3 ,,	102·2
,,	,,	,,	...	...	5 ,,	101·0
,,	,,	,,	...	...	9 ,,	102·0
,,	23	,,	...	...	9 a.m.	99·2
,,	,,	,,	...	...	9 p.m.	103·0
,,	24	,,	...	...	9 a.m.	100·0
,,	,,	,,	...	...	5 p m.	101.2
,,	,,	,,	...	...	9 ,,	102·2
,,	25	,,	...	...	9 a.m.	102·8
,,	,,	,,	...	...	5 p.m.	102·0
,,	,,	,,	...	...	9 ,,	104·8
,,	26	,,	...	...	9 a.m	102·8
,,	,,	,,	...	...	5 p.m.	101·0
,,	,,	,,	...	...	9 ,,	103·0
,,	27	,,	...	...	9 a.m.	101·8
,,	,,	,,	...	...	5 p.m.	100·9
,,	,,	,,	...	...	9 ,,	102·8
,,	28	,,	...	...	9 a.m.	101·8
,,	,,	,,	...	..	5 p.m.	101·0
,,	,,	,,	...	...	9 ,,	100·5
,,	29	,,	...	...	9 a.m.	100·8
,,	,,	,,	...	...	5 p.m.	100·5
,,	,,	,,	...	...	9 ,,	101·5
,,	30	,,	...	...	9 a.m.	102·8
,,	,,	,,	...	...	5 p.m.	101·9
,,	,,	,,	...	...	9 ,,	103·0
December	1	,,	...	...	9 a.m.	101·2
,,	,,	,,	...	...	5 p.m.	100·9
,,	,,	,,	...	...	9 ,,	103·0
,,	2	,,	...	...	9 a.m.	100·4

TABLE VII.—*M. J. Phlebitis: Septicæmia*—continued.

December 2, 1878	5 p.m.	100·9
,, ,, ,, ...	9 ,,	103·8
,, 3 ,, ...	9 a.m	101·5
,, ,, ,, ...	5 p.m.	101·0
,, ,, ,, ...	9 ,,	103·9
,, 4 ,, ...	9 a.m.	100·8
,, ,, ,, ...	5 p.m.	104·2
,, ,, ,, ...	9 ,,	103.7
,, 5 ,, ...	9 a.m.	100·6
,, ,, ,, ...	5 p.m.	104·0
,, ,, ,, ...	9 ,,	103·7
,, 6 ,, ...	9 a.m.	99·9
,, ,, ,, ...	3 p.m.	102·8
,, ,, ,, ...	5 ,,	104·6
,, ,, ,, ...	9 ,,	103·0
,, 7 ,, ...	9 a.m.	99·5
,, ,, ,, ...	5 p.m.	101·2
,, ,, ,, ...	9 ,,	103·5
,, 8 ,, ...	9 a.m.	98·6
,, ,, ,, ...	5 p.m.	98·6
,, ,, ,, ...	9 ,,	99·5

It is not my intention to enter into any speculative considerations. I wish rather to confine myself to facts. But, in comparing the extreme exacerbations and remissions met with in septicæmia, and some cases of acute phthisis, it is impossible not to ask oneself the question how far these pyrexial phenomena in the latter disease may be due to some condition analogous to that which obtains in the former.

As a contrast to the oscillations of temperature in acute phthisis, let me call your attention to those of acute capillary bronchitis. Under ordinary circumstances in bronchitis attacking the larger tubes, the temperature does not rise high; but, in capillary bronchitis, the rise is often considerable. It rarely, however, reaches the elevation of acute pneumonic, or even acute tubercular phthisis. There is, in

some cases, a difficulty in making a differential diagnosis between acute capillary bronchitis and acute phthisis. I think the thermometer affords important aid in such cases; and I have been able by its use—by recognising that the temperature did not reach a great height, although it was sustained for many days at a rather high level—to give a favourable prognosis, although the physical signs were such as might have existed in acute phthisis.

Let me call your attention to Table No. VIII, which represents the temperature-movements in a case of severe capillary bronchitis. The oscillations were very decided; but you will observe that, unlike that which occurs in phthisis, or typhoid fever, the temperature was highest in the morning—much higher indeed at 9 a.m. than at 9 p.m.; and this is not an isolated case; in several others of a like nature, I have found the temperature highest in the morning.

TABLE VIII.—*M. C., aged* 28. *Acute Capillary Bronchitis.*

Date.	Morning.	Evening.
February 7, 1877	—	101·2
,, 8 ,,	100·6	100·8
,, 9 ,,	101·5	101·7
,, 10 ,,	102·0	102·3
,, 11 ,,	103·2	99·0
,, 12 ,,	104·0	100·0
,, 13 ,,	103·0	99·0
,, 14 ,,	103·2	99·6
,, 15 ,,	102·6	99·2
,, 16 ,,	102·8	99·1
,, 17 ,,	101·6	100·0
,, 18 ,,	100·2	99·6
,, 19 ,,	98·6	99·0
,, 20 ,,	98·6	98·6
,, 21 ,,	98·0	98·6
,, 22 ,,	98·6	98·4

Table No. IX represents the temperature in the case of a woman who was admitted into the Infirmary under my care. Her condition for several days was such that I was unable to form a correct opinion of the nature of her disease. From the temperature, pulse, and general aspect, as well as the physical signs, I feared that we had to deal with a case of acute tuberculosis of the lungs. Soon, however, the temperature began to assume oscillations, which made me hope that no tubercular disease existed. The temperature was highest in the morning. Gradually the patient improved, and made a satisfactory recovery. The case turned out to be one of acute capillary bronchitis, with probably some localised pneumonia.

TABLE IX.—*E. B., aged* 25. *Acute Capillary Bronchitis.*

Date.	9 a.m.	5 p.m.	9 p.m.
November 30, 1878 ...	—	—	102·4
December 1 ,, ...	102·8	103·0	102·6
,, 2 ,, ...	102·7	—	102·6
,, 3 ,, ...	103·4	101·2	101 8
,, 4 ,, ...	100·7	102·2	103·7
,, 5 ,, ...	102·6	101·9	102·3
,, 6 ,, ...	105·8	101·4	101·4
,, 7 ,, ...	103·7	99·4	101·2
,, 8 ,, ...	103·4	102·0	101·7
,, 9 ,, ...	101·8	101·0	102·1
,, 10 ,, ...	103·4	—	99·3
,, 11 ,, ...	102·2	99·5	101·0
,, 12 ,, ...	102·0	100·5	100·2
,, 13 ,, ...	102·0	101·0	101·4
,, 14 ,, ...	102·0	99·9	100·2
,, 15 ,, ...	100·2	99·2	99·2
,, 16 ,, ...	99·5	99·2	99·9
,, 17 ,, ...	100·9	—	—
,, 18 , ...	100·2	99·5	99 5
,, 19 ,, ...	99·4	99·7	99·0
,, 20 ,, ...	99·0	98·4	99·0

The differential diagnosis between phthisis with cavities and some cases of bronchitis with dilated bronchi is by no means easy. Without attaching undue importance to thermometric observations, it cannot be doubted that, while the physical signs may be such as to admit of hesitation as to the exact nature of the affection, the thermometer may afford valuable aid. I have referred to the fact that, in capillary bronchitis, I have found that the temperature has not the afternoon or evening rise so common in phthisis; on the contrary, the temperature is highest in the morning. In the following case, which simulated in a great measure one of chronic phthisis, but which I considered to be—I believe correctly, as far as could be judged by the course which the disease took—one of bronchiectasis, I was aided much by the thermometer.

TABLE X.—*J. S. Bronchitis: Bronchiectasis.*

Date.	Morning.	Evening.
October 17, 1876	100·2	98·6
„ 18 „	99·6	99·0
„ 19 „	99·8	98·6
„ 20 „	100·4	99·3
„ 21 „	98·9	99·4
„ 22 „	100·0	98·0
„ 23 „	99·2	100·2
„ 24 „	99·0	99·5
„ 25 „	100·5	98·6
„ 26 „	100·0	98·6
„ 27 „	100·1	99·0
„ 28 „	100·6	99·2
„ 29 „	100·3	98·6
„ 30 „	99·6	99·9
„ 31 „	100 0	99·3
Nov. 1 „	99·8	98·6
„ 2 „	100·0	99·0
„ 3 „	98·6	98·6

The temperature never rose high; and, instead of having an evening exacerbation, had almost invariably an evening remission.

Very important indications of the existence of phthisis are often afforded by the use of the thermometer before any physical signs of the disease can be detected. As a remarkable instance of elevation of temperature in such a case, let me call your attention to the following.

M. C. (Table XI) was admitted into the Infirmary under my care on February 12th, 1878. She complained of having had shiverings, and of feeling generally ill. Her temperature on the day after admission was 101 deg. in the morning, and 102·4 deg. in the evening; and the following morning it had fallen to the normal. A careful examination of the patient was made, and no physical signs of disease could be detected in the lungs or elsewhere.

TABLE XI.—*M. C., aged* 40. *Phthisis.*

February 13	Morning	...	101·0
„ „	Evening	...	102·4
„ 14	9 a.m.	...	98·4
„ „	5 p.m.	...	102·8
„ „	9 p.m.	...	100·2
„ 15	9 a m.	...	101·7
„ „	1 p.m.	...	101·0
„ „	2 p.m.	...	103·8
„ „	5 p.m.	...	101·5
„ „	9 p.m.	...	99·2

There was neither cough nor expectoration. On the evening of this day the temperature rose to 102·8 deg. at five o'clock, and at nine it had fallen to

100·2 deg. The following day, at 2 p.m., it rose to 103·8 deg., and at 9 p.m. it was down to 100·2 deg.; the next day two rises occurred. These elevations of temperature were accompanied with shiverings, almost approaching to decided rigors, so that the case presented very much the aspect of an irregular quotidian ague, or one of septicæmia.

The diagnosis at which I arrived was that the case was one of phthisis; but for several days, although the chest was carefully examined from day to day, no physical signs of lung-mischief could be detected. After a time, however, slight crepitation was noticed at the right apex, and the nature of the case became clear. I considered it one of catarrhal pneumonic phthisis of a subacute form.

For some days, the temperature was irregular in its elevations and depressions; but, as the disease became pronounced, the alterations of temperature assumed a form which is, I believe, the most frequent type in phthisis—namely, that the temperature is highest at or about 5 or 6 p.m., that a fall begins from 5 to 7 p.m., that at 9 p.m. there has been a considerable fall, and that a low level is maintained during the night and forenoon.

And it is to this part of the subject that I now wish specially to call your attention.

In a large number of cases of phthisis—whether acute, subacute, or chronic—I have found *that the temperature has been highest at or near five o'clock in the afternoon; that, as a rule, there is a decided fall of temperature before nine o'clock p.m.; and that the tem-*

perature is low in the morning. There can be no doubt, I think, that there are different types as regards temperature in phthisical cases. There are some cases in which it is highest late in the evening, nine o'clock, or so. There are others in which it is highest in the morning. These are, however, exceptional cases; indeed, I think this latter kind of temperature only occurs under exceptional circumstances—circumstances of a passing character. Then, again, there are some cases of phthisis which have two exacerbations, and two remissions in the twenty-four hours, simulating, in this respect, cases of septicæmia. *But of a series of cases, now amounting to a large number, and including several thousand separate observations, the main results are as I have stated.*

I must now call your attention to a series of tables in illustration of the point to which I have referred.

TABLE XII.—*M. C, aged* 40. *Phthisis.*

Date.	9 a.m.	5 p.m.	9 p.m.
March 8, 1878	98·5	101·6	100·5
„ 9 „	100·8	—	102·0
„ 10 „	98·6	103·5	101·0
„ 11 „	98·6	101·5	100·8
„ 12 „	101 5	102·7	102·0
„ 13 „	99·0	102.8	102·0
„ 14 „	100·6	102·7	101·0
„ 15 „	99·6	101·6	100·0
„ 16 „	101·8	103·0	101·0
„ 17 „	99·7	103·8	101·6
„ 18 „	98·3	102·6	100

Table No. XII shows the temperature of the patient already referred to, as having a very high

temperature before any physical signs of lung-disease could be detected. The temperature in this case, and also in those cases which follow, was taken at 9 a.m., 5 p.m., and 9 p.m.

Table No. XIII gives the hourly variations of temperature for an entire day in the same patient.

TABLE XIII.—*Hourly Variations of Temperature in the case of M.C*

March 15, 1878	7 a.m.	97·2
,, ,, ,,	8 ,,	97·6
,, ,, ,,	9 ,,	98·0
,, ,, ,,	10 ,,	99·0
,, ,, ,,	11 ,,	99·6
,, ,, ,,	12 ,,	99·6
,, ,, ,,	1 p.m.	99·6
,, ,, ,,	2 ,,	99·7
,, ,, ,,	3 ,,	100·0
,, ,, ,,	4 ,,	100·6
,, ,, ,,	5 ,,	101·6
,, ,, ,,	6 ,,	102·0
,, ,, ,,	7 ,,	101·4
,, ,, ,,	8 ,,	100·4
,, ,, ,,	9 ,,	100·0
,, ,, ,,	10 ,,	99·8
,, ,, ,,	11 ,,	99·6
,, ,, ,,	12 ,,	98·8
March 16 ,,	1 a.m.	98·5
,, ,, ,,	2 ,,	97·4
,, ,, ,,	3 ,,	97·4
,, ,, ,,	4 ,,	98·3
,, ,, ,,	5 ,,	98·3
,, ,, ,,	6 ,,	97·6

Table No. XIV shows the temperature of a case of phthisis in the third stage (active), taken three times a day.

TABLE XIV.—*R. T., aged* 35. *Phthisis.*

Date.	9 a.m.	5 p.m.	9 p.m.
March 10, 1878	101·2	101·8	100·4
,, 11 ,, ...	100·0	102·0	101·0
,, 12 ,, ...	101·0	102.4	101·5

TABLE XIV. *R. T., aged* 35. *Phthisis*—continued.

	9 a.m.	5 p.m.	9 p.m.
March 13, 1878	101·0	102·5	101·6
„ 14 „	100·0	101·0	102·0
„ 15 „	99·2	101·6	101·0
„ 16 „	100·0	102·4	101·0
„ 17 „	102·0	102·6	101.0
„ 18 „	102·0	102·7	100·9
„ 19 „	102·6	103·0	100·2
„ 20 „	101·0	102·6	101·0
„ 21 „	102·0	102·5	101·5
„ 22 „	102·5	103·0	101·0
„ 23 „	101·0	102·5	101·7
„ 24 „	99·0	101·2	101·0

Table No. XV shows the variations of temperature in the same case for one day, at 4, 5, 6, 7, 8, and 9 p.m.

TABLE XV.—*Hourly Variations of Temperature in the case of R. T.*

March 28, 1878 ... ... 4 p.m.	100·7
„ „ „ ... ... 5 „	101·5
„ „ „ ... ... 6 „	102·0
„ „ „ ... ... 7 „	102·0
„ „ „ ... ... 8 „	101·4
„ „ „ ... ... 9 „	101·1

Table No. XVI. shows the temperature in the same patient when the disease had advanced further, when both lungs were riddled with cavities, and the patient was extremely emaciated. You will observe that the temperature was irregular. I have little doubt that, if it had been taken hourly every day, it would have been found that there was more than one exacerbation followed by remission during the day. Profuse perspirations occurred at various times—sometimes in the middle of the day; these were attended occasionally with a high temperature.

On April 20th, at 1 p.m., the temperature was 103·6 deg., and the patient was perspiring freely.

TABLE XVI.—*R. T., aged* 35. *Phthisis.*

Date.	9 a.m.	5 p.m.	9 p.m.
April 20, 1878	—	101·0	100·5
„ 21 „	100·0	101·2	102·0
„ 22 „	103·0	103·8	103·5
„ 23 „	102·0	101·0	101·9
„ 24 „	103·0	101·0	100·9
„ 25 „	100·6	99·0	99·4
„ 26 „	100·2	101·8	102·0
„ 27 „	102·5	101·0	100·3
„ 28 „	99·6	102·0	101·8
„ 29 „	102·6	101·5	101·0
„ 30 „	102·8	103·3	102·7
May 1 „	101·4	103·2	103.3
„ 2 „	102·0	103·6	103·6
„ 3 „	103·2	102·0	101·8

Table No. XVII shows the temperature in a case of phthisis running a somewhat acute course.

TABLE XVII.—*E. T. Phthisis.*

Date.	9 a.m.	5 p.m.	9 p.m.
April 24, 1878	102·0	104·0	103·2
„ 25 „	102·4	103·8	103·3
„ 26 „	100·0	103·6	102·7
„ 27 „	100·0	103·6	102·6
„ 28 „	99·0	102·4	101·3
„ 29 „	100·0	101·8	101·7
„ 30 „	101·0	103·0	102·5
May 1 „	100·8	102·7	102·8
„ 2 „	101·0	102·7	102·6
„ 3 „	101·0	103·4	102·8
„ 4 „	102·0	103·4	103·0
„ 5 „	101·4	102·5	102·6
„ 6 „	100·3	103·1	102·0
„ 7 „	99·8	102·5	102·0
„ 8 „	100·4	102.3·	102·0

Table No. XVIII shows the temperature in a case of phthisis in the third stage attended with *diuresis.*

TABLE XVIII.—*E. W. Phthisis, third stage.*

Date.	9 a.m.	5 p.m.	9 p.m.
March 25, 1878	100·0	102.4	100·0
,, 26 ,, ...	99·5	101.5	100·7
,, 27 ,, ...	100·0	102·4	101.0
,, 28 ,, ...	100·0	101·8	100·7
,, 29 ,, ...	99·5	102·0	101 5
,, 30 ,, ...	100·7	101·7	100·1
,, 31 ,, ...	99 0	102.0	101·0
April 1 ,, ...	99·5	101·8	101·2
,, 2 ,, ...	100·0	101·0	100.7
,, 3 ,, ...	99·8	102·1	101·7
,, 4 ,, ...	100·6	102·5	101·6
,, 5 ,, ...	100·9	102·0	100·9
,, 6 ,, ...	99·9	102·4	101·4
,, 7 ,, ...	100·0	101·6	100·9

Table No. XIX shows the temperature in the same case for one day, taken at 5, 6, 7, 8, 9 p.m.

TABLE XIX.—*Variations of Temperature in the case of E. W.*

April 11, 1878	5 p.m.	102·0
,, ,, ,, ...	6 ,, ...	102·0
,, ,, ,, ...	7 ,, ...	101·4
,, ,, ,, ...	8 ,, ...	100·0
,, ,, ,, ...	9 ,, ...	100·0

Table No. XX shows the temperature in a case of phthisis in the early stage.

TABLE XX.—*M. McG, aged 17. Phthisis: Early stage.*

Date.	9 a.m.	5 p.m.	9 p.m.
April 2, 1878	99·2	100·7	100·0
,, 3 ,, ...	99·1	101·1	100·6
,, 4 ,, ...	98·6	100·8	100·0
,, 5 ,, ...	99·0	101·0	100·0
,, 6 ,, ...	98·6	101·0	100·0
,, 7 ,, ...	99·0	101·0	100·0
,, 8 ,, ...	98·6	100·9	100·0
,, 9 ,, ...	99·5	101·0	100·1

But, in giving illustrations of what I have found to be the most frequent form of temperature-oscilla-

tion in phthisis, I must also add that there are many exceptions to the rule. In some cases, the temperature is highest late in the evening, about 9 o'clock, even to the extent of a degree or more; whilst, again, the usual fall which comes before that hour may be delayed, so that the temperature at 5 and 9 o'clock varies very little. (See Table No. XXI.)

TABLE XXI.—*E. M. Phthisis.*

Date.	9 a.m.	5 p.m.	9 p.m.
September 16, 1878 ...	100·2	102·0	101·7
,, 17 ,, ...	98·8	102·5	103·0
,, 18 ,, ...	99·0	102·6	103·0
,, 19 ,, ...	100·0	102·5	103·0
,, 20 ,, ...	101·0	103·0	102·9
,, 21 ,, ...	100·3	103·0	103·2
,, 22 ,, ...	99·0	103·0	103·1
,, 23 ,, ...	100·8	102·6	102·0

I have already mentioned that in some cases more than one exacerbation followed by remission occurs. It is chiefly, I believe, in the later stages of the disease that this takes place. Table No. XXII shows the variations of temperature (in a case of phthisis in the third stage) during one day, taken every third hour. In this case, there were two distinct exacerbations.

TABLE XXII.—*Variations of Temperature. A. M. Phthisis.*

February 8	...	...	6 a.m.	...	...	...	101·0
,, ,,	...	...	9 ,,	...	...	...	101·6
,, ,,	...	...	12 ,,	...	...	...	99·0
,, ,,	...	...	3 p.m.	...	...	...	97·2
,, ,,	...	...	6 ,,	...	...	...	100·0
,, ,,	...	...	9 ,,	...	...	...	103·0
,, ,,	...	...	12 ,,	...	...	...	100·5
,, 9	...	...	3 a.m.	...	...	...	99·8
,, ,,	...	...	6 ,,	...	...	...	100·5

As the result of these observations, it would appear that, whether phthisis is in an early or a late stage, if active mischief is going on, there are, as a rule, important variations of temperature, and that in a majority of cases—I believe in a very large majority—the highest temperature is marked about or near 5 o'clock p.m., and that there is a very decided remission in the morning. I need not insist on the importance of this fact in reference to the diagnosis of phthisis, and on the great error we might commit in estimating the condition of a patient by a single thermometric observation, made, for instance, in the morning in our own consultation rooms, just at the time when the temperature is lowest. No temperature observations can be relied on in forming a diagnosis in a case of incipient phthisis, unless made at least twice or thrice a day; and, if only one observation is possible, it should not be made in the morning.

I cannot stop to consider at any length the question as to how far phthisis may be progressive without there being any elevation of temperature. It is quite certain that some cases are met with in which there is little or no elevation, even where the disease is advanced, but these are exceptional.

In considering the facts which I have brought under your notice, one cannot help asking whether this particular form of temperature-oscillation is characteristic of phthisis only, or whether it may not be so of all forms of allied diseases. I have endea-

voured to ascertain what is the usual course in other forms of tuberculous affections, as of the bones, peritoneum, etc.; but at present my facts are not sufficient to warrant me in coming to any definite conclusion, and I must wait for further inquiries. I will, however, refer to some cases which have a bearing on the subject.

In lumbar abscess and caries of the bones, I have found a more elevated temperature in the evening than in the morning. In one case of pelvic abscess with diseased bone, the temperature was for a few days taken at 9 a.m., 5 p.m., and 9 p.m., and it was always highest at 5 p.m.

In a very interesting case of peritoneal inflammation, which was for a long time very obscure as to its real nature, the observation of the temperature led me to infer that there was some tuberculous condition. The most careful examination of the lungs revealed no disease, and the physical signs observed in the abdomen were such as might be consistent with the formation of a tumour. In the progress of the case, the temperature assumed the type which I have referred to as existing so often in phthisis; and I was led to express an opinion that there was some tuberculous condition present. The temperature-oscillations are represented in Table No. XXIII. The patient died, and the necropsy revealed the existence of tubercular peritonitis, and a few tubercles were found in the lungs.

TABLE XXIII.—*M. C., aged* 28. *Tubercular Peritonitis: a few Tubercles in the Lungs.*

Date.	9 a.m.	5 p.m.	9 p.m.
April 26, 1878	99·0	102·0	102·6
,, 27 ,, ...	101·0	102·0	101·6
,, 28 ,, ...	98·6	103·0	102·3
,, 29 ,, ...	98 6	102·0	101·9
,, 30 ,, ...	100·3	103·0	102 5
May 1 ,, ...	99·8	104·0	102·8
,, 2 ,, ...	99·0	104·6	103·9
,, 3 ,, ...	99·4	103·6	101·3
,, 4 ,, ...	98·6	103·7	102·0
,, 5 ,, ...	98·3	103·0	101·8
,, 6 ,, ...	98·3	102·6	102·0
,, 7 ,, ...	99·0	102·0	101·3
,, 8 ,, ...	98·6	101·4	100·6
,, 9 ,, ...	99·3	101·0	100·8
,, 10 ,, ...	99·0	100·8	100·8
,, 11 ,, ...	98·3	100·3	100·0

In considering these temperature-oscillations, it is impossible not to note that, to a certain extent, they seem to be exaggerations of normal changes. In the healthy, there is a fall in the evening and early morning. So in phthisis; but, then, in the healthy, the late morning temperature is fairly high, whilst in phthisis it is often very low.

But I must now pass on to consider some other points. I need not dwell on the importance of the use of the thermometer in the early days, and throughout the whole course, of typhoid fever. There are, however, several points in connection with the temperature of that disease to which I should like to call your attention, but time forbids. One subject I will refer to, viz., how far the pyrexia of typhoid resembles that of acute phthisis. It is well known that there are consider-

able oscillations in the temperature of typhoid, the evening temperature being higher than that of the morning; and, indeed, towards the end of an attack, there is a very marked morning defervescence, almost amounting to an intermission. But, in the early days of the fever, this does not obtain.

It has been usual with me, of late, whilst having the temperature of my fever cases taken frequently, to have a record of it made three times a day, as in phthisis, viz., at 9 a.m., 5 p.m., and 9 p.m. My numbers are not yet enough to enable me to speak decidedly on the point we are considering; but I have found that, as a rule, the temperature is higher at 9 p.m. than at 5 p.m.; but this is not always so. In some cases, the temperature has been highest at 5 p.m.

Amongst the many interesting questions in connection with our subject is: What is the highest temperature which is consistent with the maintenance of life in the human economy? All observations hitherto made tend to show that, when the temperature of the body in any case of pyrexia reaches eight or ten degrees above the normal, there is grave danger unless the heat is speedily reduced. Wünderlich says that the highest recorded temperature in a living man was 112·55 deg. Fahr. Brodie found the temperature 111 deg. in a case of injury of the spinal cord. But this temperature was far surpassed by that which is said to have occurred in the remarkable case recorded by Mr. Teale, of Scarborough, in which a lady, who suffered an injury

whilst riding is reported to have had a temperature which on one occasion reached 122 deg.; and Mr. Teale says that, during seven weeks, the temperature never fell below 108 deg., and rarely below 110 deg., and yet the patient recovered.

This case is so remarkable and so exceptional that I think we may set it aside as having no applicability to ordinary cases of fever; and we must, I think, in spite of it, conclude that a temperature of 110 to 112 deg. is not compatible with anything like a prolonged maintenance of life.

Cases of intermittent fever sometimes have a very high temperature. I have myself known a rise to over 107 deg.; but Dr. Macintosh states that he has known a rise to 110 deg.; but, then, this high temperature soon subsides, and a very rapid defervescence takes place.

In pneumonia, the temperature rarely rises above 106 deg. in the axilla. In a series of 151 cases of the acute form of the disease, referred to in another chapter, which have occurred in my hospital practice, there is only one case, with the exception mentioned below, in which the temperature reached 106 deg. in the axilla, and it was not fatal. In only a small proportion of cases did the temperature (taken in the axilla) pass beyond 105 deg. In one case, complicated with rheumatic fever, the temperature rose to 108·8. It subsequently fell, but the patient ultimately died. The highest recorded temperatures in cases ending favourably that I am acquainted with are 106·7 deg. (Ziemssen) and

107 deg. (Kocher). In fatal cases, a temperature of 108·9 deg. to 109·4 deg. has been reached before death.

An examination of the temperature-charts of cases of acute pneumonia affords interesting evidence of the nature of the pneumonic fever. It runs, in some cases, a very definite course; a high temperature is maintained for some time, a so-called crisis comes, and there is a very rapid defervescence. But this is by no means always the case. There are often two or three decided remissions, followed by exacerbations, before the final defervescence comes. Tables No. XXIV and XXV illustrate these points.

TABLE XXIV.—*R. T., aged* 17. *Pneumonia.*

Date.	Morning.	Evening.
January 17, 1872	—	106
,, 18 ,, ...	104·8	104·3
,, 19 ,, ...	104·3	105·4
,, 20 ,, ...	102·0	99·5
,, 21 ,, ...	97·5	98·0
,, 22 ,, ...	99·4	99·8
,, 23 ,, ...	99·3	99·5
,, 24 ,, ...	99·5	—
,, 25 ,, ...	99·0	—
,, 26 ,, ...	98·4	—

TABLE XXV.—*J. W., aged* 14. *Pneumonia.*

Date.	9 a.m.	5 p.m.	9 p.m.
April 10, 1878	—	105·0	103·7
,, 11 ,, ...	101·6	102 0	102·8
,, 12 ,, ...	104·0	104·0	102·0
,, 13 ,, ...	100·5	103·0*	104·0
,, 14 ,, ...	99·0	—	98·6
,, 15 ,, ...	98·0	98·6	98·6
,, 16 ,, ...	99·0	98·6	99·0

* Same temperature at 1 p.m.

In cases of septicæmia, the temperature has risen to 109·6 deg., and yet life has been maintained; but apparently this has resulted from the fact that the heat has been quickly subdued by the external application of cold. Again, in typhoid fever, when the temperature has risen to an alarming height, to over 107 deg., a rapid cooling has followed the use of the tepid bath, and life has been apparently thereby saved.

In injuries and diseases of the nervous system, a very high temperature has been recorded. I have referred to Brodie's and Teale's cases. But, in nervous diseases, I have never found, in my own experience, that life has been maintained when a temperature such as would be probably fatal in other diseases has been reached. Table No. XXVI shows the temperature of a man who was the subject of cerebral hæmorrhage, and also of a slight fracture of the internal table of the skull. He was admitted into the Infirmary under my care with apoplectic symptoms. For several days, the temperature was low,; but, during the last three days of life, it rose considerably, and reached, shortly before death, 107·4 deg.

TABLE XXVI.— ——. *Cerebral Hæmorrhage.*

Date.	Morning.	Evening.
November 11, 1877	98·6	98·6
„ 12 „	99·0	98·6
„ 13 „	100·0	98.0
„ 14 „	98·6	98·6
„ 15 „	99·0	99·0
„ 16 „	98·6	99·0

TABLE XXVI.— —— *Cerebral Hæmorrage*—continued.

	Morning.	Evening.
November 17, 1877	98·6	99·0
,, 18 ,, ...	99·0	99·0
,, 19 ,, ...	98·6	99·0
,, 20 ,, ...	98·6	99·0
,, 21 ,, ...	100·0	103·0
,, 22 ,, ...	103·0	103·5
,, 23 ,, ...	105·0	107·0

Shortly before death, 107·4

Table No. XXVII shows the temperature-ranges in a case of delirium tremens complicated with pneumonia. The first rise indicates the pneumonic fever from which the patient partially recovered; but the delirium continued, and, before death, there was a decided rise of temperature, reaching 107·4 deg. shortly before dissolution.

TABLE XXVII.—*S.S., aged* 32. *Delirium Tremens, with Pneumonia.*

Date.	Morning.	Evening.
November 7, 1877	—	98·6
,, 8 ,, ...	99·0	—
,, 9 ,, ...	100·8	99·7
,, 10 ,, ...	104·0	101·0
,, 11 ,, ...	101·0	100·4
,, 12 ,, ...	100·0	100·0
,, 13 ,, ...	99·0	100·7
,, 14 ,, ...	99·6	100·0
,, 15 ,, ...	99·0	100·0
,, 16 ,, ...	99·0	100·0
,, 17 ,, ...	99 0	101·0
,, 18 ,, ...	—	101·0
,, 19 ,, ...	99·0	100·0
,, 20 ,, ...	99·2	—
,, 21 ,, ...	102·4	104·5
,, 22 ,, ...	107·4	—

It would be very interesting, if time permitted, to consider the causes of increased temperature in

pyrexia; but, perhaps, in the present uncertainty of our knowledge, it would not be very profitable. Although increased heat constitutes one of the most prominent symptoms of fever, it is assuredly only one of its factors, or one of its results; and yet it is impossible to shut our eyes to the great importance of carefully attending to it as an indication for treatment. When we see, in cases of septicæmia, of acute rheumatism, of typhoid fever, etc., the temperature rising to 107, 108, 109, or 110 deg.; and when, apparently as the direct result of this increased heat, we witness the gravest disturbances of the functions of the nervous centres, and dissolution threatened; and when, under the influence of external cold, we see the temperature rapidly fall, the coma disappear, the delirium subside, and the patient restored to a condition of comparative comfort and real safety, it is impossible not to come to the conclusion that this high temperature, whatever may be its essential nature, has been, in these cases, a main cause of the alarming symptoms which were present. Were the coma or the delirium due in either case to the poisoned or altered condition of the blood, the mere reduction of heat would scarcely afford the marked relief which we witness. Were the phenomena the result of some impression on the nervous centres produced by organic change, however slight, it is difficult to understand how this lowering of temperature could give rise to the rapid and striking alterations which are sometimes witnessed; and seeing that tempera-

ture plays so important a part in the phenomena of the pyrexial state, we may well, in studying the therapeutics of fever, give a large share of our attention to those measures which have the effect of diminishing heat. This part of the subject is one on which I cannot enter now, although I hope on some future occasion to lay the results of my observations before the Society, and to speak of my experience of the use of cold applied externally, of quinine, of alcohol, of digitalis, of aconite, of salicylic acid, and of free nutrition as temperature-depressors in various forms of fever.

I may here remark, however, that in the treatment of typhoid fever, and of hyperpyrexia in acute rheumatism, I have employed very largely the tepid bath, and tepid and cold sponging, with apparently very great advantage. My practice in typhoid fever is to give directions that the temperature be taken at frequent intervals, every hour, or every two hours, and, should it rise to 104 deg. Fahr., that the patient be sponged thoroughly well with tepid or cold water. The effect of this, when well done, is usually to lower the temperature, and as soon as it rises again the sponging is to be renewed. Should the temperature, in spite of the sponging, go on rising and reach 105 deg. or 106 deg., I always advise that the tepid bath should be used. The temperature of the bath at first should be about 95 deg., and the heat should be gradually reduced by the addition of cold water. I may say in brief that I have no confidence in the use of large doses

of quinine for the permanent reduction of temperature in typhoid fever, but I am in the habit of prescribing the drug to the extent of fifteen or twenty grains, in divided doses during the day, in cases where the temperature is persistently high.

In estimating the rise and fall of temperature in disease, it is obvious that we cannot trust to our unaided senses; but that, in the thermometer, we possess a guide which, if properly used, can scarcely deceive us. The rapid rises of temperature which sometimes occur can only be recognised and appreciated by the application of this instrument of precision; and it cannot be doubted that it has in many cases rendered most valuable service, and been the means of saving life by pointing out the necessity for prompt and decided action.

CHAPTER II.

Pneumonia—(Clinical Lecture).

Gentlemen,

I purpose in the present lecture to lay before you an analysis of a series of cases of acute pneumonia, which have occurred to me in my hospital practice. I have not included in the series cases which have been treated by me in private practice, either alone or in conjunction with other practitioners, for the reason that such cases are not open to public criticism, and frequently cannot be observed with the same care and accuracy as are practicable with cases occurring in hospital. The experience now referred to extends over a period during which I have been physician to two large hospitals—first, the Liverpool Northern Hospital; and, secondly, that in which we are now assembled.* Into the former hospital, a large number of seamen are admitted; and many of the cases of pneumonia which came under my care there occurred in men who were presumably in good health previous to the attack, which had come on suddenly, after exposure to cold, whilst the men were doing their ordinary work on board ship. I mention this fact as, I think, it has a bearing on the question of treatment, to which I shall have to refer.

* The Liverpool Royal Infirmary.

The cases number one hundred and fifty-one. They are drawn up in the table which I show you,* which presents the leading features of each case—viz.: the initials, the age, sex, occupation, and previous health, as far as could be ascertained, of the patient; the date of the commencement of the attack, generally marked by the occurrence of rigor; the date of admission into hospital; the side and the extent of lung involved; the frequency of the pulse; the number of the respirations; the temperature, when taken by the thermometer; the treatment; the date of convalescence *i.e.*, of the period when all active symptoms had subsided, when the pulse had fallen to a natural or nearly natural standard, 60 to 80, when the temperature was normal, and the patient able to take solid food. The table further includes the number of days during which the patient had been under treatment when convalescence was established, and the number of days which had elapsed from the beginning of the attack; the date of discharge, and the number of days the patient was in the hospital; and, lastly, the result, with remarks on the complications of the case, if there were any.

As to the age of the patients—under 10 years of age there were five cases; between 10 and 20 years, twenty-three cases; between 20 and 30, fifty-six cases; between 30 and 40, thirty-seven cases; between 40 and 50, twenty cases; between 50 and 60, eight cases; between 60 and 70, two cases. One

* See page 179.

hundred and twenty patients were males; thirty-one were females. Many of the patients were strong, robust-looking men, in whom the disease had only existed for a few days before admission into hospital, the attack being distinctly traceable to exposure to wet or cold, or to both.

The list includes men engaged in a great variety of occupations. There were seamen, porters, hawkers, stewards, tailors, etc.; one is described as a gentleman.

Of the one hundred and fifty-one cases, the pneumonia was single in one hundred and twenty-one, and double in twenty-four. In six cases the site of the disease is not noted. Of the single cases the right lung was the seat of the disease in fifty-seven; the left in sixty-three; in one case the side is not mentioned. Of the double cases the right lung was most involved in six, the left in ten; both lungs were equally involved in seven cases. In one case there is no record on this point. In twelve of the single cases the pneumonia was seated in the apex and upper lobe of the lung, and in eight of these the disease was on the right side. In eleven cases the pneumonia was complicated with bronchitis; in twenty-one there was pleurisy; in two there were symptoms of gangrene of the lung—one of these recovered. Nine cases were connected with acute rheumatism. In five of these there was peri- or endo-carditis, or both.

Of the one hundred and fifty-one cases eleven died, viz., Nos. 7, 55, 81, 82, 84, 109, 118, 131, 132,

138, and 141, giving a rate of mortality of 7·28 per cent. No. 7 died from the result of sudden and extensive effusion into the pleura, after convalescence from the pneumonia had set in. No. 55 was admitted into the hospital in a sinking state, and died within forty-eight hours after admission, hepatisation of the whole of the right lung being found after death. No. 81 had evidence of previous phthisis, and a cavity was found in one lung at the necropsy. No. 82 was a woman of worn-out constitution, who sank from excessive diarrhœa. No. 84 had gangrene. No. 109 was admitted in a state of exhaustion, and died within two days. No. 118 was a case of double pneumonia occurring in a very intemperate man, who died on the third day after admission into the Infirmary. No. 131 was also a very intemperate man; he died four days after admission, and tubercles were found in one lung. No. 132 was also intemperate. No. 138 was the case of a woman admitted with rheumatic fever, and who had hyperpyrexia, the temperature rising to 108·8 deg. No. 141 died within two days after admission.

With reference to the one hundred and forty cases which recovered. In 125 the date of convalescence is recorded. In nine cases convalescence was established at the end of the third day of treatment; in fifteen at the end of the fourth day; in twenty at the end of the fifth day; in fifteen at the end of the sixth day; in thirteen at the end of the seventh day; in twelve at the end of the eighth day; in eight at the end of the ninth day; in six at

the end of the tenth day; in four at the end of the eleventh day; in one at the end of the twelfth day; in six at the end of the thirteenth day; in three at the end of the fourteenth day; in one case at the end of the fifteenth day; in five cases at the end of the sixteenth day; in one case at the end of the nineteenth day; in one at the end of the twentieth day; in one at the end of the twenty-second day; in one at the end of the twenty-third day; in two cases at the end of the twenty-fourth day; in one case at the end of the twenty-eighth day. In fifteen cases the date of convalescence is not noted.

The average duration of these 125 cases, from the commencement of treatment to the period of convalescence, was 8·21 days. But it is also important to ascertain how soon convalescence was established from the commencement of the attack, as well as from the commencement of treatment.

In eighty-six of the cases in which the date of convalescence is noted, I was able to ascertain the date of the commencement of the attack. Of these one case was convalescent at the end of the fifth day of the attack; nine at the end of the sixth day; nine at the end of the seventh day; seven at the end of the eighth day; six at the end of the ninth day; five at the end of the tenth day; fifteen at the end of the eleventh day; eight at the end of the twelfth day; five at the end of the thirteenth day; five at the end of the fourteenth day; four at the end of the fifteenth day; two at the end of the sixteenth day; two at the end of the seventeenth day;

four at the end of the nineteenth day; one at the end of the twenty-second day; one at the end of the twenty-fifth day; one at the end of the twenty-sixth day; and one at the end of the thirty-first day. This gives an average of 11 days for the 86 cases. In the remaining cases, where the date of convalescence is recorded, that of the commencement of the attack could not be accurately ascertained.

In taking the average of the number of days during which the patients were in the hospital, it must be borne in mind that for the most part they were kept in, not only until they had fully recovered from their attack, but had also gained sufficient strength to be able to resume work. Further, out of the one hundred and forty cases which recovered, thirteen remained in the hospital several weeks—more than fifty days—in consequence of other causes besides the pneumonia, such as acute rheumatism, heart complications, acute and chronic dysentery, &c. In six cases, one of which after recovering from the pneumonia died of heart disease, the date of discharge from the hospital is not noted. Excluding these latter, and the thirteen cases of very protracted stay in hospital, we have for the remaining 121 an average of 24·44 days for each case from the date of admission to the date of discharge.

The result of these cases, especially when taken in conjunction with others of which statistics have been given by other authors, tends to show that pneumonia, under the present mode of treatment, is by no means a fatal malady; and that, at least

in the ordinary run of hospital cases, a moderate mortality may be hoped for.

Before I proceed to make any remarks on the treatment adopted in these cases, I wish to make a few observations on some of the conditions which characterise pneumonia.

First, as regards the pulse. I do not wish to refer to its frequency, but to its character. Speaking generally, the pulse in pneumonia, although in many cases apparently full, is wanting in firmness, and indicates a feeble arterial tension; in fact, it is essentially dicrotic, and this dicrotism has been more or less marked in all the cases in which I have taken sphygmographic tracings of it. I show you tracings taken from four patients.

Figs. 1 and 2 were taken from John W. (No. 79 of the series), a healthy-looking man, twenty years of age, who was admitted on May 15th, 1873, with pneumonia of the left lung. The pulse was 90, full, but soft and compressible. The tracing Fig. 1

Fig. 1.

was taken on the day after admission, being the fifth of the attack. You see it is essentially dicrotic. On the 19th the pulse was 56, and tracing Fig. 2 was taken. You observe that the dicrotism is only slightly marked; that, in fact, the pulse had become almost normal in its character.

Fig. 2.

Tracing Fig. 3 was taken from James W. twenty-eight years of age (No. 77), who was suffering from right pleuropneumonia in the early stage. The attack, which was preceded by symptoms of congestion of the liver, commenced on the 22nd of April, 1873, and the tracing was taken on the 23rd. It presents well-marked dicrotism. The patient was treated with carbonate of ammonia, and six ounces of brandy daily. He was convalescent on the sixth day.

Fig. 3.

Tracing Fig. 4 was taken from a woman (No. 123 of the series) who was suffering from severe double pneumonia. The tracing was taken the day after her admission into the infirmary, about the fourth day of the attack. She was treated with carbonate of ammonia, cinchona, and six ounces of brandy daily. She was convalescent four days after admission.

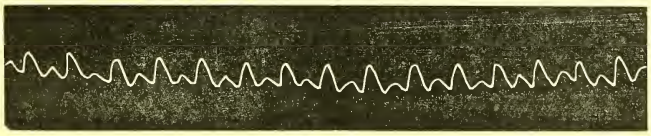


Fig. 4.

Tracing Fig. 5 shows extreme dicrotism of the pulse. It was taken from a patient who was suffering from tubercular pneumonia. The man lingered for several weeks; he was admitted on the 5th April, and died on the 16th May. He presented all the features of that adynamic form of pneumonia which is associated with tubercle. After death we found both lungs studded with tubercles; the right solidified, with small cavities in the lower lobe; the left in a state of engorgement. The dicrotism of the pulse was perceptible to the touch. The tracing was taken on the 23rd April. Stimulants and nourishment were given freely throughout the attack, but the character of the pulse did not alter except to become quicker and weaker as the case progressed.

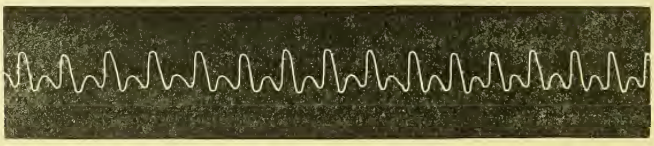


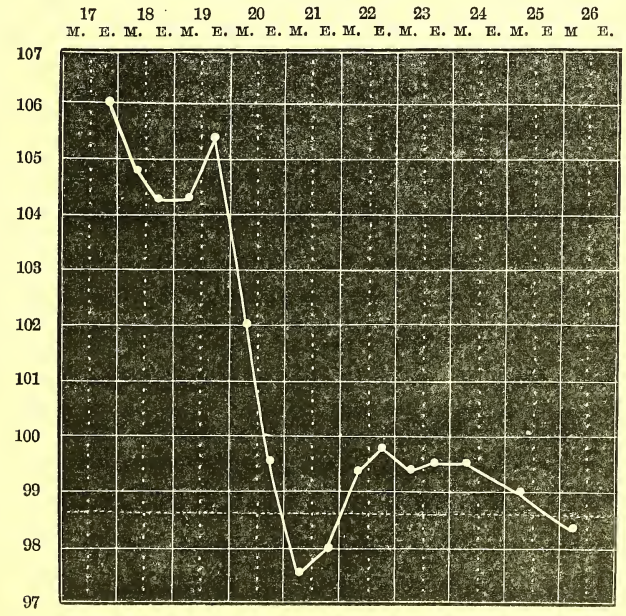
Fig. 5.

The comparison of these tracings with each other is, I think, very instructive, and may show you that, should you be in doubt in any case, the sphygmograph may possibly give you valuable assistance.

As regards temperature. Before the introduction of the clinical thermometer into general use, the importance of observing the frequency of the pulse and of the respiration was insisted on, but now we

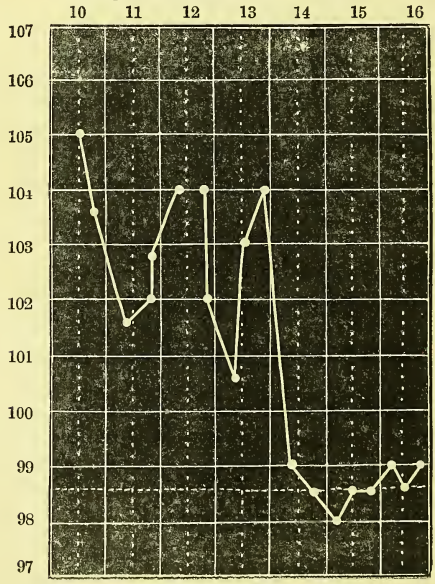
also insist on the observation of the temperature; and this affords a most valuable guide as to the progress of a case. As a rule, it may be said that, in acute pneumonia, the temperature rises rapidly and reaches a high level in the early period of the attack; that for the most part the oscillations of temperature during the acute stage are not very great; and that there are no marked remissions, as is the case in typhoid fever and acute phthisis. But there is a feature in the temperature of acute pneumonia which is very striking, viz., its rapid fall when what we may term the crisis arrives. I show you the temperature-chart (Fig. 6, Robert T.) of a case of very acute pneumonia in which this rapid

Fig. 6.—*Robert T., January*, 1872.



defervescence is well marked. From the evening of the 19th January to the evening of the 20th, viz., within twenty-four hours, the temperature had fallen six degrees; from 105·4 to 99·4; and on the following morning it was below the normal. Now, although as a general rule, whenever the temperature makes a decided fall in pneumonia, we may consider the case is practically over, it would be very rash to come to this conclusion in all cases. Indeed, we occasionally find that there is more than one well-marked fall, with subsequent rises, before the final defervescence occurs. The chart (Fig. 7, John W.) which I now show you illustrates this point very well.

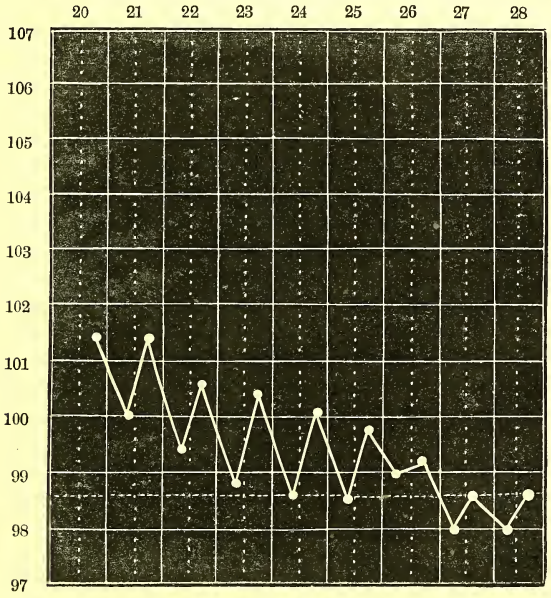
Fig. 7.—*John W., April*, 1878.



When three records are given, the temperature was taken at 9 a.m., 5 p.m., and 9 p.m. On April 13th, the temperature was also observed at 1 p.m., when it was at the same height as at 5 p.m.

Occasionally the temperature falls gradually from day to day, and then nothing approaching to a crisis occurs. The defervescence is more like that which takes place in typhoid fever. This is illustrated in the following chart (Fig. 8), which was taken from a woman suffering from inflammation of the right lung (No. 140 of the series).

Fig. 8.—*Ellen M., February*, 1886.



The temperature in pneumonia reaches a high level in some cases. It rarely, however, rises above 106 deg. in the axilla. In the series of cases referred to in the table where the temperature was taken, there is only one instance, with the exception referred to below, in which it reached that level, and in only a small proportion of cases did it pass

beyond 105 deg. The highest recorded temperatures in cases ending favourably that I am acquainted with are 106·7 deg. (Ziemssen) and 107 deg. (Kocher). In fatal cases a temperature of 108·9 deg. and 109·4 deg. has been reached before death. In some of the worst cases of pneumonia, those which assume a typhoid character, the temperature maintains a somewhat low level—102 deg. to 104 deg.

In No. 138 of the series the temperature rose on one occasion to 108·8 deg. This was a case of acute rheumatism, with double pneumonia. The external application of cold reduced the temperature, which never rose again to an alarming height. The patient ultimately succumbed. The high temperature in this instance must, I think, be considered as due to the rheumatic condition.

The occurrence of a prolonged and well-marked rigor as an initial symptom of pneumonia is a well-known fact. In the series of cases, it was very distinctly ascertained to have existed in a large number, but in others its existence could not be satisfactorily made out.

Some cases of pneumonia are ushered in with severe gastric disturbance, bilious vomiting, etc.; whilst, in a few, the disease is preceded by a distinct attack of jaundice.

Although pneumonia undoubtedly has a tendency to terminate favourably after a certain number of days, by rapid defervescence—by so-called crisis—yet the duration of cases differs very materially,

depending on a variety of causes; and no precise rule can be laid down with reference to the time any given case will last.

The table shows that of the cases that recovered the largest number were convalescent at the end of the fifth day of treatment, viz., twenty; then there were fifteen convalescent at the end of the fourth day; fifteen at the end of the sixth day; thirteen at the end of the seventh day; and eleven at the end of the eighth day, &c.

The table also shows that of the eighty-six cases in which the date of the commencement of the attack was clearly ascertained, convalescence set in, in the largest number, viz., sixteen, at the end of the eleventh day of the attack, in ten at the end of the sixth day, in eight at the end of the seventh day, and in eight at the end of the twelfth day, &c.

In twelve of the cases in the table, the pneumonia was situated in the apex and upper lobe of the lung. These cases of apex-pneumonia often present features of great interest. I have seen several of them, and they have been marked, especially when occurring in women, by profound disturbance of the nervous system—delirium of a most violent kind, sometimes assuming a maniacal character. Usually such cases do well, but they always give occasion for great anxiety, for a pneumonia of the apex is usually an indication of one of two conditions, either of the presence of some tubercular deposit, or else of a depraved and vitiated constitution. I shall refer later on to this subject of apex-pneumonia.

I now proceed to speak briefly of the treatment which was adopted in these cases, and as my object is simply to describe to you my own practice, and to give you the results of it, I shall refrain from any remarks on the various measures which you may find recommended by others.

In no instance was venesection practised. Only three patients were cupped, viz., Nos. 2, 7, and 9. In only three cases were leeches applied, viz., Nos. 3, 5, and 7. In Nos. 1 and 13, leeches had been applied before admission.

In twenty cases only—twenty of the early numbers of the series—antimony was given in small doses varying from one-sixteenth of a grain to a quarter of a grain, except in two cases, Nos. 2 and 5, in which it was given in doses of a grain and three-quarters of a grain respectively.

Calomel was not given in any case. In one case, No. 10, I gave blue pill twice a day for six days, but no soreness of the gums was produced. In no other instance was mercury given except as a purgative, in combination with some other drug, either at the commencement, or during the course of the treatment. Aconite was never given.

In a large majority of the cases—in nearly 80 per cent.—carbonate of ammonia was given from the beginning of treatment and throughout the attack. This was combined with cascarilla, cinchona or senega, and often with spirits of chloroform.

In a large majority of cases some alcoholic stimulant—wine or brandy, more frequently the latter—

was given early in the disease, usually from the beginning of treatment, and was continued throughout the attack. The stimulants were given at regular intervals, every hour, or every two, three, or four hours, frequently with food—beef-tea or milk. In the most severe cases, brandy was given every hour, or hour and a half. The quantity varied; it rarely exceeded six ounces daily. In a few instances, it was eight ounces, and in three, twelve ounces. In some of the milder cases, no alcoholic stimulants were given, carbonate of ammonia and spirits of chloroform with cascarilla being alone used.

In some cases, quinine was given; and I wish to observe that I consider quinine in rather full doses a very valuable therapeutic agent in some cases of pneumonia. In two cases of the series, it was given to the extent of sixteen and twenty grains daily, and a marked improvement followed its exhibition. Both these were cases in which the pulse reached 140; one was that of a pregnant woman, who aborted during the attack. I shall refer again shortly to the question of administering quinine in the disease.

Mild counter-irritation was always resorted to; large mustard and linseed-meal poultices were for the most part applied throughout the attacks; blisters were never used in the acute stage, and only occasionally during convalescence, when the matters effused into the lung were being slowly absorbed.

In every case, nutrients were allowed freely, viz., beef-tea and milk from the beginning of treatment, and solid food as soon as the patients could take it.

With reference to the value of quinine in pneumonia. The cases which, in my opinion, are most benefited by this drug are those which occur in debilitated constitutions, where the pulse is quick and there is a persistent high temperature—cases which do not come to the crisis early, and in which the defervescence is gradual.

In the following case, quinine appeared to act beneficially, and I quote it as an illustration.

Eliz. B., aged thirty-seven, a laundress, was admitted into No. 15 ward, on December 12th, 1877. She had crossed from the Isle of Man on the 7th, being then in good health. She was exposed to wet and cold during the passage, and on the day after her arrival she had got very wet. On the 10th she had a shivering fit, pain in the side and cough. On the 13th, the day after her admission into hospital, when I saw her, there was dulness with tubular breathing over the lower three-fourths of the left lung. The pulse was 140, the respirations were 36, and the temperature was 104 deg. The sputa were rust-coloured and tenacious. The urine was free from albumen. She was ordered five grains of carbonate of ammonia, with cascarilla and spirits of chloroform every four hours, and four ounces of brandy daily, with milk and beef-tea. From this day the symptoms increased in severity, and the dulness gradually increased in extent until it involved the whole of the left lung. The pulse remained very rapid, and the temperature high. Diarrhœa had also set in, which made me fear that perhaps the case was one of typhoid fever; but there were no spots. There was delirium at night, and altogether the case looked very unpromising. On the 17th, the pulse was 136, respirations 52, temperature 104 deg., and the whole of the left lung was solidified. The brandy had been increased to

six ounces daily, and I now ordered the following mixture :— Quiniæ sulph., gr. xvj; acid sulph. dil., ʒfs.; sp. chlorof., ʒij; aq. menth. pip. ad. ℥vj; ℥j 4tis horis. On the 18th, I ordered the mixture to be given every three hours, thus increasing the quinine to twenty grains in the day. The temperature now began to fall, as you will see by looking at the chart (Fig. 9), and the pulse became less frequent. On the 21st, I reduced the quinine to sixteen grains during the day, and on the 24th, the temperature having nearly touched 99 deg., I further reduced it to ten grains during the day, and the brandy to five ounces. The diarrhœa continued for several days, but gradually subsided without special treatment. On the 26th December the brandy was stopped, and eight ounces of port wine were given. On the 28th a mixture of iron and quinine was ordered in the place of the quinine, and subsequently, during her recovery, the patient took hypophosphite of soda, and cod liver oil. For several weeks she remained very ill, but a steady, although slow, improvement had set in. She remained in the Infirmary till April 18th, and then went to the Convalescent Hospital at Woolton.

In the very young and the very old pneumonia is very fatal, but with regard to children who have passed extreme infancy my experience, chiefly gathered from private practice, is that they respond very satisfactorily to treatment. I do not think that children are at all bad subjects for pneumonia. The treatment I have adopted has been similar to that which I have pursued in adults. I generally give aromatic spirits of ammonia and small quantities of brandy in proportion to the age of the child. I also use mustard and linseed poultices, and I allow any simple kind of nourishment which the child can take.

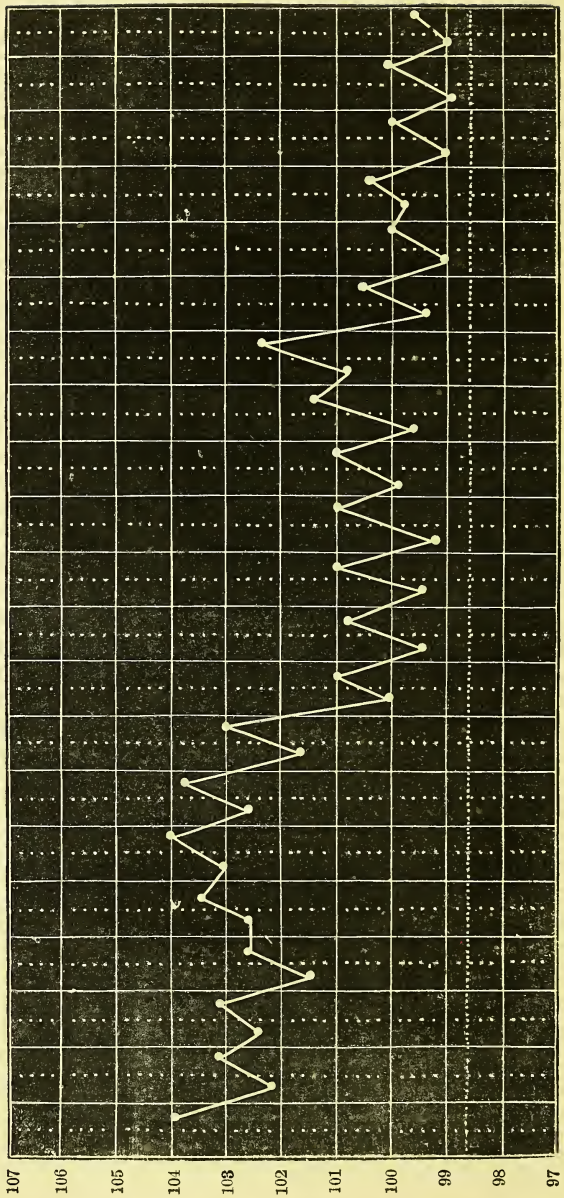


Fig. 9.—*E. B., December*, 1877.

I never use measures such as would be likely to depress the circulation, or weaken the action of the heart.

With regard to the aged. I have seen cases recover when patients had nearly reached the age of eighty years; but there cannot be a doubt that, although in childhood and adult life, pneumonia is not a very fatal malady, yet after adult life the danger of an attack considerably increases with increased years.

The advantages of early treatment are very marked in pneumonia. In hospital practice we frequently see cases which have been neglected for some weeks in their own homes. In these the consolidation of the lung remains, although all active symptoms, fever, &c., have subsided. In such cases, the process of absorption of the effused matters is often very slow, whereas, in all probability, if the patients had come early under treatment, rapid recovery would have taken place.

A few words on the subject of so-called "septic pneumonia." I have not been able to satisfy myself that there is any special form of pneumonia to which this term may be properly applied. Pneumonia is undoubtedly modified by the conditions of each patient, by his surroundings, and his constitutional state. If a patient, who is inhabiting a house which is permeated by sewer gas, contracts a pneumonia, the disease assumes characters which may lead to the attack being considered "septic," but the modifications are due, not to any special charac-

ter of the disease, but to the influences which the inhalation of the sewer gas has had on the individual. Again, I have seen cases of pneumonia occurring after confinement or abortion which have presented the usual characteristics of the disease, modified more or less by the special condition of the patient.

I have promised to say a few words about apex pneumonia, and I think I cannot do better than refer briefly to two cases which illustrate very well the profound nervous disturbances with which such cases are at times marked.

Case 1.—*Pneumonia of Right Apex.—Recovery.*—Mrs. S., aged forty-one, had, on Nov. 24th, 1877, well marked rigors, followed by an attack of broncho-pneumonia. On the night of the 27th she had delirium, and I saw her in consultation on the following day. The pulse was 112, the temperature 105 deg. Fahr. There was some dyspnœa; crepitant and subcrepitant mucous râles were heard over the chest, but there was no decided dulness on percussion. The finest râles were heard over the left front. The expectoration was rust-coloured.

She was ordered five grains of carbonate of ammonia every three hours, and six oz. of brandy during the day. On the 29th, the pulse was 112, and the temperature 102·4 deg. On the 30th, there had been violent delirium during the night; the pulse went up to 150. I saw the patient at 11 a.m. She had then the aspect of a person suffering from delirium tremens. There was fine crepitation at the left apex; the pulse was 108, and the temperature normal. The urine was examined, and found free from albumen. Bromide of potassium was ordered to be taken in twenty grain doses every four hours, the dose to be increased if the delirium persisted. The ammonia and brandy were continued.

Dec. 1. Had had three slightly convulsive attacks. Was ordered half a drachm of bromide of potassium every four hours. At 2.45 p.m. the pulse was 96, and the aspect better, but there was a wild look about the patient. There was dulness over the upper part of the left lung, with crepitation, but no dulness below. The brandy, ammonia, and bromide were continued.

Dec. 4. The pneumonia had now subsided. There had been delirium, with hallucinations, the patient praying and singing at times. The eyes were prominent and bright, and there was great restlessness. The brandy was diminished, and some claret was given. Food was being taken.

On the 6th, the patient was quite well, with the exception of the mental aberration, which continued, but there had been sleep. On the 10th, she was much better and calmer with fewer hallucinations; in fact, the nervous symptoms had almost entirely disappeared. The brandy had been stopped. She recovered perfectly.

Case 2.—*Pneumonia of Right Apex.—Recovery.*—Mrs. B., aged thirty-two, was seized with "a bilious attack," on the 25th Sept., 1875, and was seen on the 26th by her medical attendant. I saw her on the 29th, at 6.30 p.m. The pulse was 120, and the temperature 105 deg. The face was flushed. There was some slight crepitation over the right lung; the bowels were relaxed from purgatives. On the 30th, at 6.30 p.m., the pulse was 136, and the temperature 105·3 deg. The physical signs were the same as on the previous day. On Oct. 1st, at 5.30 p.m., the pulse was 120, and the temperature 105 deg. The bowels were still loose. There were subcrepitant râles at the back of the right lung, and the sputa were slightly rust-coloured. Five grains of carbonate of ammonia were ordered to be taken every four hours, and turpentine stupes to be applied to the chest.

On Oct. 2, there was dulness on percussion over the

right apex, and fine crepitation was heard over the same region. The patient's manner was excited. The pulse was 126, and the temperature 103·8 deg. Ordered to take two teaspoonfuls of brandy every hour, and to continue the ammonia.

On Oct. 3, the patient had had a bad night, followed by violent delirium and excitement in the morning. She took her brandy and food well. Small doses of chloral hydrate were added to each dose of the ammonia mixture, after which she had some sleep. At six p.m., the pulse was 120, and the temperature 103·8 deg. She looked calmer and better. There were coarse râles, almost simulating gurgling, under the right clavicle, and crepitation at the apex behind. She had been taking the ammonia and chloral every three hours.

On Oct. 4, at 6.30 p.m., the pulse was 86, and the temperature normal. She was quiet, and had slept almost constantly since my last visit. The ammonia was ordered to be taken every fourth hour, and five ounces of brandy during the day.

On Oct. 7, the patient was well. The pulse was 80, and the temperature normal. There had been no delirium since the 4th. The only remnant of the lung affection was the presence of some dulness at the right apex with crepitation which was less coarse than that noted on the 4th.

I did not see her again. She perfectly recovered.

CHAPTER III.

Bronchitis and other Diseases of the Bronchial Tubes.*

1. Acute Bronchitis—2. Chronic Bronchitis—3. Plastic Bronchitis—4. Dilatation of the Bronchial Tubes—Bronchiectasis—5. Narrowing or Obstruction of the Bronchial Tubes—6. Cancer of the Bronchial Tubes.

1. *Acute Bronchitis—Acute Bronchial Catarrh.*

Definition.—An acute inflammation or congestion, general or partial, of the bronchial tubes.

Ætiology.—The causes of acute bronchitis may be classed as (*a*) *predisposing* and (*b*) *exciting*.

(*a*) Of the *predisposing* causes age is one of the most important. The disease is indeed confined to no period of life, but it is most frequently met with in the young and the old, and in these subjects it assumes its most serious characters. The imperfect development of the infant, and the diminished vitality of the aged, seem to render them especially liable to attacks of bronchitis, and to make the disease exceptionally fatal in them. Sex appears to have no influence as a predisposing cause. The habits of life have an important influence in the causation of bronchitis. The practice of living in heated rooms, especially where gas is largely consumed, and of breathing the vitiated atmosphere

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produced by the assemblage of large numbers of persons in apartments, is undoubtedly a fertile predisposing, as well as exciting, cause of the complaint; so also is the practice of keeping children too much within doors on the one hand, or, on the other, of exposing them to inclement weather when insufficiently clad. Temperament can scarcely be considered a predisposing cause, but the state of the general health exercises a powerful influence. A weakly constitution, or one weakened by over-work, improper food, &c., predisposes to bronchitis; whilst such affections as Bright's disease, gout, and diseases of the heart, alike favour its occurrence. Again, certain occupations are favourable to the development of bronchitis. Independently of the fact that living or working in heated and ill-ventilated rooms predisposes to the disease, such occupations as lead to the inhalation of irritating particles, as those of steel, cotton, &c., give rise to it. The climate most favourable to the production of bronchitis is probably that which is at the same time both cold and damp, and where sudden variations of temperature occur. The seasons of the year in which it prevails most are the late autumn, the winter, and the early spring.

(*b*) *Exciting causes.*—Although undoubtedly cold directly applied to the surface of the body is, in a large number of cases, the exciting cause of bronchial inflammation, still the transition from cold to heat—passing from a cold atmosphere to a heated one—is a large factor of the disease. There can be little

doubt that bronchitis is often produced directly by the effects of heated and vitiated air on the bronchial membrane, and on the system at large; and that in the latter instance, the affection is merely a local manifestation of a general influence. Bronchitis may also be caused by the direct action of irritants contained in the air—as irritant vapours, minute particles of steel, cotton, or ipecacuanha, and the emanations (pollen) from flowering plants. Again, morbid conditions of the blood, the result of specific febrile affections, act as exciting causes of the disease; as do also the poison of syphilis, and the altered condition of the blood produced by gout. Bronchitis is, moreover, a constant accompaniment of influenza.

Anatomical Characters.—The mucous membrane is mainly affected in acute bronchitis, but morbid changes may be produced in the deeper structures. The mucous membrane is red—the redness being arborescent, streaked, or mottled, but not usually spread uniformly over a large surface. The injected condition of the membrane does not, as a rule, extend into the finer bronchial tubes, but in some cases where there have been frequent attacks of inflammation, the smallest bronchi have a red appearance. The membrane is sometimes thickened and soft, but ulceration is very rare. The tubes are generally found more or less filled with secretion, either frothy mucus, muco-pus, or even actual pus. Sometimes the secretion is very abundant, filling all the tubes. Fibrinous masses are occasionally met

with, which may form casts of the tubes. Collapse of portions of lung-substance—lobulettes or whole lobules of the lungs—is not unfrequently found, as are also patches of lobular pneumonia. The venous system and the right side of the heart are overloaded, and the blood is dark. In many cases fibrinous deposits are found in the cavities and great vessels of the heart.

In speaking of the pathology of bronchitis, it is necessary to refer to the distribution of the bronchial blood-vessels. The bronchial arteries when they have fairly entered the lungs have no accompanying veins. The so-called bronchial veins are some small vessels which return the blood supplied to the structures about the roots of the lungs. The blood which is supplied to the bronchial tubes, when they have commenced their divisions, passes into radicles of pulmonary veins, and is returned directly to the left side of the heart. The question whether there is a communication between the bronchial arteries and the pulmonary artery, is still *sub judice*. If such communication exist, it is only slight. The blood of the bronchial arteries, after supplying the mucous membrane and other structures of the tubes, passes, either wholly or in very large part, to the left side of the heart, not having circulated through the aërating portion of the lungs.* The circumstances of this anatomical arrangement are most important in a practical point of view. Anything which

* For further consideration of this subject the author's work on the Anatomy of the Human Lung may be referred to.

embarrasses the circulation on the left side of the heart—such as mitral regurgitation—must necessarily cause a very loaded condition of the bronchial vessels; and all physicians are familiar with the form of bronchitis which is so common in these cardiac affections. The congested mucous membrane, and the profuse bronchial secretion, are the result of the direct impediment to its circulation which the blood meets with, from passing at once into vessels which go straight to the left side of the heart. The relief often afforded in this form of bronchitis by the exhibition of digitalis, is explained by the circumstance above referred to.

Symptoms—The symptoms of acute bronchitis vary according as the larger or smaller tubes are affected. The disease attacks, first, the larger and medium-sized tubes; and, secondly, the smaller ones. To this latter form of affection the name of *capillary bronchitis* has been given.

1. Acute bronchitis of the *larger tubes*. The attack is usually ushered in by symptoms of catarrh—sneezing, lachrymation, a sense of fulness about the nose and eyes, with frontal headache; the throat becomes dry and sore, and then increased secretion sets in; the follicles at the back of the pharynx become enlarged; the upper part of the larynx is often involved, there being slight hoarseness; and the affection gradually creeps down into the bronchial tubes. The disease is not ushered in by decided rigors, but chills and sometimes shiverings are experienced; the pulse is not much affected,

but its frequency is increased in some cases; there is a general sense of malaise, as well as a want of energy. When the disease has set in fully certain local symptoms are found. More or less pain is felt behind and above the sternum; the sensation is increased by a deep inspiration; the pain shoots at times over the chest in the direction of the larger bronchial tubes; and there is a tickling or unpleasant irritation felt behind the sternum, which gives rise to cough. Dyspnœa is not a marked feature of this form of bronchitis; it exists, however, sometimes; and in the most severe cases a sense of oppression, weight, and tightness about the chest is experienced. Cough is one of the earliest and most prominent symptoms; it is at first dry, and there is usually at this period some hoarseness. The cough is paroxysmal, and often very violent; it becomes attended with expectoration as the disease progresses. This varies at different stages of the affection; at first watery and frothy, and almost transparent, it becomes as the disease progresses more consistent, viscid, and opaque, passing through the stages of mucus, to muco-pus, and pus; it is sometimes distinctly nummulated. Small streaks of blood are occasionally seen mixed with the sputa. Examined under the microscope the sputa are found in the early stages of the disease to contain epithelial cells from the mucous membrane; and, later, many of the so-called exudation-corpuscles, molecular and granular matter, pus-cells, and occasionally blood-discs.

In the milder cases of this form of bronchitis there is but little general disturbance; and even in the more severe cases the febrile reaction is not usually very great. The pulse rises a little, but does not become very frequent; the temperature rarely becomes high; there is in many cases but little interference with the appetite. A general feeling of depression, which in some cases is very marked, is usually experienced.

2. Acute bronchitis of the *smaller tubes—Capillary bronchitis.* This is a very formidable disease. It attacks the finer bronchial tubes, and probably extends to their smallest ramifications. Its symptoms are very grave. Some of the worst cases of capillary bronchitis are met with in connection with emphysema of the lungs. It may be an extension of inflammation from the larger tubes; or the capillary tubes may be attacked simultaneously with the larger ones, or alone. The early symptoms are more severe than those of ordinary bronchitis, and rigors are more common. Dyspnœa is marked; it may vary from mere rapid respiration to constant or paroxysmal orthopnœa. The respirations may rise to fifty in a minute. Cough is almost continuous, at times becoming very violent and most distressing. Expectoration is attended with difficulty. The sputa soon become very abundant, and rapidly assume a purulent character; or they are very viscid and ropy.

The general symptoms are very severe. The fever is high—the temperature reaching to 103 deg. Fahr.

and upwards; and the pulse is frequent, rising to 120 or 140. The temperature rarely attains the height which characterises acute tuberculosis or pneumonia. There are often profuse perspirations, and in some cases excessive debility is felt. If the disease progresses unfavourably, symptoms of very imperfect aëration of the blood come on. The face becomes turgid and bloated, the lips and ears get livid, the veins are distended, the temperature falls, cold clammy perspirations break out, the pulse becomes very small and rapid, delirium supervenes, the respiration is shallow and catching, and the patient dies of apnœa, and from the presence of fibrinous clots in the heart and great blood-vessels.

Physical Signs.—The physical signs of both forms of acute bronchitis may be referred to together. Inspection reveals little of practical value in simple bronchitis. The chest-form is not altered. In severe cases the abdominal movements are in excess. The costal movements are frequently those of elevation rather than expansion. In extreme cases the lower end of the sternum and the connected cartilages sink with inspiration; while the expiration movements are slow, laboured, and inefficient. If the hand is applied to the chest, rhonchal fremitus may be often felt, sometimes over a large area. The percussion-sound may be somewhat exaggerated from over-distension of the lungs, especially in children; or not appreciably altered; or deficient in resonance, owing to the accumulation of secretion at the bases of the lungs, to œdema or congestion

(as in typhoid fever), or to pulmonary collapse. In young children a sound resembling the cracked-pot sound may be occasionally produced, variable in site. The sounds heard in auscultation vary according to the stage of the disease. The breath-sounds are loud when the tubes are free; when the latter are plugged by secretion, they often become feeble or even totally suppressed, from closure of a tube leading to a portion of the lung. The adventitious sounds of bronchitis include the various rhonchi, dry or moist: the dry rhonchi are heard in the early stages of the disease for the most part, but when once secretion has set in, the moist rhonchi, or râles, are more or less extensively heard, depending for their character on the size of the tubes which are the seat of inflammation. Thus they are called *mucous* when produced in the large tubes, *sub-mucous* and *sub-crepitant* when produced in the finer ones; the latter term being used to characterise the râles of capillary bronchitis. When the large bronchial tubes are filled with a secretion which is not viscid, the sounds may have a rattling character. The various rhonchi may be heard over different parts of the lungs at the same time, according to the seat and stage of the bronchitis. In capillary bronchitis sub-crepitant râles, accompanying inspiration and expiration, are abundantly heard, towards the bases of both lungs especially. As a rule there is no displacement of organs in bronchitis, but the diaphragm is sometimes depressed from great distension of the lungs, and

the heart is occasionally displaced towards the right.

Diagnosis.—The diagnosis of acute bronchitis, except in a few instances, presents no great difficulty. In the early stages of whooping-cough it is impossible to decide whether the case is one of simple bronchitis or not, but subsequently the paroxysmal character of the cough settles the point. In some cases of bronchitis occurring in children the breathing may resemble that of croup, but here the presence of catarrh; the wheezing nature of the respiration; the absence of much fever; the characters of the sputa obtained by wiping the back of the tongue, and its freedom from membranous shreds; and the physical examination of the chest indicating the presence of rhonchi, will be sufficient to establish a diagnosis. From laryngitis the discrimination is not difficult.

The diagnosis of pneumonia from capillary bronchitis, with which form of bronchitis the former disease can alone perhaps be confounded, is generally easy. Capillary bronchitis is not ushered in, as pneumonia usually is, by a well-marked and prolonged rigor; the general febrile disturbance is less, and the temperature not so high; moreover the absence of dulness on percussion, and of increased vocal resonance and fremitus will aid in the differentiation. From lobular pneumonia in children the diagnosis is not always easy. In this disease there is often no dulness to be perceived on percussion; whilst, on the other hand,

in bronchitis dulness may exist from pulmonary collapse.

The diagnosis of capillary bronchitis from acute phthisis often presents difficulties. The main points to be relied on, independently of the family history, which may aid, are that in capillary bronchitis the fever is less and the temperature lower; signs of apnœa soon come on; and there is free expectoration of muco-purulent matter. Further, the oscillations of the temperature have for the most part a different character, a point which I have referred to in a preceding chapter. In one form of acute phthisis there is evidence of pneumonic consolidation, followed by signs of the formation of cavities. In the miliary tubercular form there are in many cases scarcely any physical signs except râles, most marked at the apices of the lungs.

Prognosis, Duration, Termination, and Mortality.—The prognosis in an ordinary case of bronchitis is favourable, but when the disease occurs in the very young or the aged the prognosis should always be guarded. In the milder forms the affection may last only a few days, or two or three weeks. Severe cases are more protracted. The disease may terminate in perfect recovery, in death, or by passing into the chronic form. It may be the starting point of emphysema of the lungs, or of certain forms of phthisis. The mortality is much influenced (1), by age, being greatest in the very young and the very old; (2), by the previous state of heath, which, if lowered by any circumstances, will render recovery

more doubtful; (3), by the extent of the inflammation, especially when the disease is of the capillary form; (4), by the existence or non-existence of any organic disease of the heart, lungs, or kidneys; (5), by the disease being epidemic or otherwise; and, lastly, by the time at which the case has come under treatment, whether early or late.

Treatment.—In the treatment of bronchitis regard must be had to the constitutional condition of the patient. Care must be taken to ascertain whether the disease is secondary to some organic affection; or the result of mechanical irritation, of the presence of gout or rheumatism in the system, or of influenza; or whether it arises idiopathically. The treatment of the disease as a primary affection will be considered first.

In an ordinary case of acute bronchitis it is very desirable to keep the patient confined to his room and, if the case is at all severe, to his bed. The temperature of the apartment should be maintained at from 60 deg. to 65 deg. Fahr. A higher temperature than this is generally not favourable to the progress of the case. In the early stages of the attack it is well to allow the air of the room to be more or less saturated with steam. A free action of the skin should be promoted; and for this purpose warm drinks, with or without some form of alcohol or some diaphoretic medicine, may be given; or a hot-air bath may be used in bed. Great relief is often experienced from the application of a large mustard or mustard and linseed-meal poultice to

the chest; and it is well, if mustard is applied first, to apply immediately afterwards a large hot linseed-meal poultice, to be renewed every few hours. This constant application of warmth and moisture to the chest is often productive of very great relief to the symptoms.

Cases of acute bronchitis do not require venesection, nor is the application of leeches often, even if ever, called for. Severe counter-irritation is moreover to be prohibited. It is generally desirable to act on the bowels, and a mercurial, followed by a saline purgative, will often be of great service. In the old and debilitated, as also in the young, all lowering treatment must, however, be avoided. In the early stages of the affection, before secretion has commenced, and when the mucous membrane is dry and the cough hard, diaphoretics with ipecacuanha may often be given with advantage; but as soon as secretion is fairly established, carbonate of ammonia, spirits of chloroform, ether, cascarilla, senega, or such-like drugs should be administered. Indeed in almost every stage of bronchitis carbonate of ammonia is one of the most valuable remedies we possess. Care should be exercised, especially with the aged, that nothing should be given which will so nauseate as to prevent food being taken. In the exhibition of medicines to alleviate the cough, regard must be had to the condition of the patient and the stage of the disease. Opium in all its forms should be given with caution, especially in the young and old. It no doubt often succeeds in

checking cough, but in doing so it also checks expectoration, and causes an accumulation in the bronchial tubes, which sometimes becomes very dangerous to life. Chloral in small doses is often of great use for relieving cough, and it may be combined with oxymel of squills. It has also a good effect in allaying spasm of the tubes, if this exist. In some cases of bronchitis the question of procuring sleep becomes an important one. Opium in its various forms is generally inadmissible, in consequence of its tendency to increase the condition of apnœa; but chloral may be given with safety, and the recovery of a patient may sometimes be dated from the sleep which this agent procures.

In reference to the exhibition of alcholic stimulants, except in the early stages, and in certain cases dependent on a gouty or rheumatic condition, they should usually be given in smaller or larger quantities. They increase expectorating power, and ward off the tendency to apnœa. In the old they are especially called for, and, together with carbonate of ammonia, should form the main therapeutic agents to be relied on. In the treatment of capillary bronchitis, ammonia and alcholic stimulants should be exhibited from the commencement, and the quantity must depend on the symptoms of each case. There is one source of danger in capillary bronchitis which should always be borne in mind, viz., the formation of fibrinous clots in the heart and great blood-vessels. These deposits become the proximate cause of death in many cases, and they are especially

liable to form when there is emphysema of the lungs. Their presence may often be correctly inferred during life from the respiration becoming very rapid, shallow, and laboured: the pulse being quick, weak, and small, although the heart may at the same time be felt beating vigorously; the voice becoming feeble; and the mental faculties seriously impaired. After death a large portion of the cavities of the heart may be found occupied by these deposits, the calibre of the pulmonary artery and aorta being also materially diminished by them.

In many cases of bronchitis, when the acute symptoms have passed off but the secretion continues profuse, as well as in those cases called bronchorrhœa, the exhibition of iron is often of great service. It seems to give tone to the relaxed capillaries of the mucous membrane, and to diminish the secretion. It may be given in combination with carbonate of ammonia, in the form of the ammonio-citrate; or the tincture of the perchloride with ether or spirits of chloroform may be employed, or the ethereal tincture of the acetate (Ger. Ph.), which is a very valuable preparation in some cases.

Inhalations are useful for allaying cough in the earlier stages of the affection, or for the relief of spasm. In some cases of severe bronchitis where apnœa has been threatened, recovery has followed the exhibition of large doses—half an ounce—of turpentine. In this dose, however, it sometimes produces alarming symptoms, and it is perhaps better to exhibit it in smaller quantities tentatively.

An emetic may be serviceable, especially in children, if the tubes are much loaded. Children suffering from severe attacks of bronchitis should not be allowed to sleep long, for fear of dangerous accumulation in the tubes, and care should be taken that the secretions do not collect about the back of the mouth.

Patients should not be kept on a low diet even at the beginning of an attack, and as the disease progresses the quantity of food allowed may be increased according to the appetite. In the treatment of gouty bronchitis, or bronchitis associated with a tendency to the formation of uric acid in the system, colchicum and the alkalies must be given, and the general measures used which are applicable to the constitutional condition. If bronchitis depend on a gouty state, it will not yield to the ordinary treatment, but when its cause is recognised and the appropriate remedies are administered, the symptoms usually soon begin to improve.

In the cases of bronchitis which are connected with heart disease, and especially with mitral regurgitation, digitalis is often of great value. By steadying the action of the heart it relieves the overloaded pulmonary veins, and thus directly diminishes the congestion of the mucous membrane, as mentioned in the paragraph relating to the pathology of the disease.

It is impossible in the scope of this article to refer specially to the treatment of bronchitic attacks arising from the various kinds of mechanical irrita-

tion. There is, however, one form of bronchitis which may be mentioned, viz., that connected with hay-fever, arising either from the inhalation of pollen, or caused by some peculiar atmospheric influence acting on a peculiar nervous system. It is very difficult of cure. In the writer's experience no remedies seem to have any particular influence over it, and it is usually only to be relieved by removing the patient from the exciting cause of the affection. In the treatment of bronchitis depending on constitutional syphilis, the appropriate measures for that affection must be resorted to.

2. *Chronic Bronchitis—Chronic Bronchial Catarrh.*

Definition.—A chronic inflammation or congestion, more or less extensive, of the bronchial tubes.

Ætiology.—Chronic bronchitis very frequently results from repeated attacks of the acute disease, but it may be of a chronic character from the beginning. Emphysema of the lungs, dilated bronchi, and phthisis are causes of the complaint; as are also various forms of heart disease, and some blood affections, such as gout. The inhalation of irritating particles gives rise to chronic bronchitis; and it is also met with in connection with chronic alcholism. It is most common amongst the old.

Anatomical Characters.—The bronchial mucous membrane is discoloured, being of a dull red tint, greyish, or brownish. The discoloration is for the most part partially, but sometimes evenly, diffused. There is swelling and increased firmness of the

mucous membrane, and the sub-mucous tissue in old-standing cases becomes infiltrated and indurated. The fibrous and muscular tissues are hypertrophied; the cartilages in the larger tubes are sometimes calcareous; and there is generally more or less emphysema of the lungs.

Symptoms.—The symptoms of chronic bronchitis vary greatly in different cases. They resemble in kind those of the acute affection. There is cough, expectoration, pain, soreness or uneasiness behind the sternum, with more or less dyspnœa. The constitutional symptoms may be very slight, scarcely any effect on the general health being apparent; or they may be very severe. Three forms of chronic bronchitis are recognised clinically:—1. That which includes the ordinary cases of the disease, varying much in severity; 2. that characterised by excessive secretion—*bronchorrhœa*; 3. that form which is called *dry catarrh*.

1. In the first form of chronic bronchitis the cough is at first slight, perhaps only occurring during the winter, being altogether absent in the summer. After a time the attacks become more frequent and at last the patient is never free from the affection, which is aggravated at times. The cough in such cases is more or less severe, but usually most so in the morning. It is often paroxysmal, and sometimes very violent. The expectoration, in some cases being scanty, viscid, and difficult to discharge, is in others, especially old-standing cases, copious and easy. The sputa vary much both

in appearance and quantity. They may be yellowish-white muco-purulent matter, or more decidedly purulent, of a greenish-yellow or bright or dark green colour; they are but little aërated, sometimes not at all, so that they sink in water: at times they are nummulated and quite opaque. In some cases the expectoration is fœtid, constituting the form of the disease denominated "fœtid bronchitis," the odour resulting either from sloughs of minute portions of the mucous membrane, or from chemical changes taking place in the sputa. Occasionally streaks of blood are met with. Microscopically the sputa are found to consist of epithelium, pus-cells, and granular matter, with at times blood-corpuscles.

The constitution does not suffer much in mild attacks, but when chronic bronchitis is permanent and general, the system at large sympathises more or less severely: the appetite fails, sleep is disturbed by the cough, emaciation sets in and sometimes becomes marked, but it does not proceed beyond a certain point, unlike that of phthisis, which is usually progressive. In all cases of chronic bronchitis there is great risk of an acute attack coming on, especially amongst the aged. These attacks are very dangerous, in consequence of the rapid extension of the disease throughout the lungs, and its asphyxiating character.

2. The second class of cases is characterised by excessive secretion from the bronchial tubes—*Bronchorrhœa.* This form is often met with in the old and feeble, and especially in cases of valvular

disease of the heart. The cough is paroxysmal, and attended with the expectoration of a large quantity of thin watery glairy fluid, or of thick ropy gluey matter, like white of egg. The quantity expectorated is sometimes very large. This form of bronchitis may cause death somewhat suddenly by apnœa. During the paroxysms of cough there is dyspnœa, but at other times it is absent, except when heart disease exists. The constitution suffers little, and the flux seems sometimes to be beneficial in cases of obstructive cardiac disease.

3. The third variety, or *Catarrhe sec*, is characterised by very troublesome cough, oppression of breathing, tightness of the chest, and sometimes severe dyspnœa. Expectoration is either absent or very scanty, the sputa consisting of small masses of tough viscid semi-transparent mucus. There is usually no febrile disturbance. The disease is met with in gouty people, and is often associated with emphysema of the lungs. Pathologically it seems to consist in a congested condition of the bronchial tubes.

Physical Signs. — Inspection reveals nothing abnormal in the form or size of the chest, unless emphysema of the lungs is present. The expansion in long-standing cases is usually deficient; the chest being raised more than in health. Expiration is often prolonged. Rhonchal fremitus may be felt more or less, depending on the state of the bronchial tubes. There is often increased resonance, from the presence of emphysema. The breath-sounds are

more or less changed; they are harsh and loud, and the expiration is prolonged in cases that have existed for a considerable period. The rhonchi vary; they are dry, coarse, moist, or bubbling according to the condition and contents of the tubes. Vocal resonance varies; it may be bronchophonic, normal, or deficient.

Diagnosis.—There is usually but little difficulty in the diagnosis of chronic bronchitis. The affection is most likely to be confounded with phthisis, but the character and degree of the wasting, and the absence of increased temperature, of hæmoptysis, and of the physical signs of consolidation, will generally enable the practitioner to decide in favour of the less important disease. The main difficulty lies in the diagnosis of cases where the bronchial tubes are dilated; this will be referred to hereafter.

Prognosis.—Although in itself not a dangerous malady, chronic bronchitis becomes so in consequence of the liability which exists to the occurrence of acute symptoms; when once established in middle or advanced age it is almost incurable. The complaint is further serious from its tendency to produce emphysema and dilatation of the bronchial tubes. *Per se* it can scarcely ever be said to kill.

Treatment.—No case of chronic bronchitis can be successfully treated without due regard to the constitutional condition of the patient. In some cases it is impossible to cure the disease, and all efforts should be directed towards preventing its extension; alleviating the symptoms to which it

gives rise; and warding off acute attacks. Attention must be especially paid to the state of the heart and kidneys; the duration of the affection; the age of the patient; the characters of the expectoration; the state of the lungs, as to the existence of emphysema or other morbid conditions; and the presence of gout or rheumatism. Speaking generally, chronic bronchitis must be treated by the use of a generous diet, with more or less stimulants; by the exhibition of expectorants and tonics; and by the avoidance of all depressing measures. The function of the liver must be looked to, and the administration of a few doses of blue pill with a saline aperient often gives great relief, and alters the character of the bronchial secretion. If gout, or a tendency to the formation of uric acid, is present, colchicum with alkalies and other remedies for gout, such as a course of Friedrichshall or Carlsbad waters, will prove of great service. If cardiac disease exist, whether in the form of valvular incompetence, or of weak, flabby, or dilated heart, digitalis combined with iron frequently produces marked benefit.

In the treatment of ordinary cases of chronic bronchitis, not dependent on any organic disease or constitutional condition, the patient's general health has to be looked to. The affection has a tendency to lower the health and to diminish strength, and therefore the various tonics may often be given with great benefit. Of these the most useful are quinine, the preparations of iron, and those of zinc. Cod-liver oil is also very valuable in some cases where

there is much wasting. The cases of bronchitis marked by excessive secretion are generally best treated by tonics ; whilst those where the secretion is slight—cases of "dry catarrh"—being often associated with a gouty condition of the system, are more amenable to the use of colchicum, the alkalies, iodide of potassium, and mineral waters.

In what may be called the symptomatic treatment of the affection, the various expectorants are useful—carbonate of ammonia, ipecacuanha, squill, cascarilla, senega, chloroform, &c., and these may often be beneficially combined with some form of tonic. In many cases of chronic bronchitis the expectorating power is diminished, and stimulating expectorants are of great service. Great caution must be exercised in the administration of opiates and other narcotics or sedatives. When, however, the mucous membrane is very irritable, and when there is but little secretion with a troublesome cough, these remedies are indicated. Opium is of great value, and chloral-hydrate is also very useful, as well as, in some cases, henbane and hydrocyanic acid, or, whenever spasm is present, stramonium, lobelia, the ethers, and cannabis indica. Inhalations are sometimes very beneficial, as of the vapour of warm water, iodine, creasote, and other substances. The inhalation of creasote is especially valuable if the expectoration is fœtid.

Counter-irritation is one of the most important means we possess of relieving chronic bronchitis. The irritation should not be excessive, but should be

long-continued. Iodine over a large surface of the chest, so as to keep up a constant slight inflammation of the skin, is perhaps the best application that can be used; but other irritants may be tried, such as sinapisms, or the various stimulating liniments.

The general management of the patient is most important. A mild climate should, if possible, be chosen in the winter. The patient should live as far as possible in an atmosphere which is mild and dry. Although some cases are benefited by a moist and warm atmosphere, the majority of cases of chronic bronchitis do better in a drier one. The skin must be carefully looked to, its action should be well maintained, and warm clothing always worn. A moderate amount of some alcoholic stimulant is generally desirable; and the food should be nutritious and of easy digestion. Relief will often be found from wearing a respirator.

3. *Plastic Bronchitis.*

This is a rare form of disease, and of its particular causes nothing is known. It is perhaps connected with some peculiar diathesis. Sir John Rose Cormack suggests that it may be a variety of diphtheria. It may occur in either sex, and at any period of life, but is most frequent in those who are of a strumous or phthisical constitution. It has been known, however, to attack persons of apparently healthy frame and in the enjoyment of robust health.

Anatomical Characters.—Plastic bronchitis is

anatomically characterised by the formation of concretions in the bronchial tubes. These concretions consist of fibrinous exudation from the mucous membrane; they form casts of the tubes, and are expectorated. These casts are either solid or hollow, and on examination are always found to consist of concentric laminæ. They are, for the most part, poured out into the finer bronchial tubes, sometimes, however, into the larger ones, but never into the trachea. The casts are of a whitish colour, but they are often stained with blood. Microscopically they consist of an amorphous or fibrillar material, with exudation-corpuscles, granular matter, and oil-globules.

Symptoms.—This disease is essentially chronic, but it has been met with as an acute affection in children. At the times when the casts of the tubes are expelled exacerbations occur, the patient being attacked with pain and a sense of constriction across the chest, dyspnœa, and an irritating cough. After a time, varying from some hours to a few days, the dyspnœa becomes very urgent, and the cough very severe; then, after a paroxysm of coughing, it is found that the patient has expectorated some solid material, either with or without blood, usually intermixed with ordinary bronchitic sputa. The dyspnœa and cough now subside, to recur after an interval of a few hours or longer. The disease may last for weeks, months, or even years, marked from time to time by severe accessions, and relieved by the expulsion of further concretions. The matter expelled is

often in small masses, but at times casts of bronchial tubes with several ramifications are expectorated. The disease may recur at intervals for many years; the general health in such cases does not seem to suffer, the breathing during the intervals being unaffected. There is, in some instances, an absence of febrile symptoms during the attack, whilst in others the fever is more marked. With the general symptoms are combined the *physical signs*. These are somewhat peculiar. The bronchial tubes being obstructed, portions of lung are deprived of air; the breath-sounds are therefore faint or absent. There may be dulness more or less complete on percussion, from collapse of the lung-substance, or, as occurs in some cases, from localised pneumonia. Apnœa may be threatened if a large tube is blocked up.

Diagnosis.—The diagnosis of this affection turns on the peculiarity of the expectorated matters. Doubtless the disease may be mistaken for ordinary bronchitis or pneumonia, but when once the fibrinous casts of the tubes are observed in the sputa, the nature of the case becomes clear.

Prognosis, Duration, Terminations, and Mortality.—The prognosis, if the disease is uncomplicated, is favourable; but there is great liability to recurrence. The complaint may last for many years; and may terminate in complete recovery. A fatal result generally depends on the presence of some other organic disease, such as phthisis or pneumonia.

Treatment.—But little can be advanced as to the value of any special treatment for this affection. Iodide of potassium is said to have been employed with success. The chief object should be to maintain the general health by hygienic measures, and the exhibition of tonics, such as iron, quinine, and cod-liver oil, especially if there be any tubercular taint. During the exacerbations the administration of ammonia and the use of inhalants should be resorted to, and the general principles on which ordinary bronchitis is treated should be carried out.

4. *Dilatation of the Bronchial Tubes—Bronchiectasis.*

This is a rare disease, which arises as a secondary affection. It is often associated with serious pulmonary mischief, and is at times difficult of diagnosis. There are two forms of bronchiectasis, namely, *general or uniform dilatation;* and *saccular or ampullary dilatation.*

1. The *general* or uniform bronchiectasis consists in a cylindrical dilatation of one or more of the tubes throughout a considerable portion of their extent. The tubes are evenly widened for the most part, and end abruptly.

2. The *saccular* form of bronchiectasis consists of a globular dilatation of a tube at one point or at several points. The dilatations vary in size, being from half an inch to an inch or more in diameter. On the tracheal side they usually communicate with a slightly enlarged bronchial tube, whilst on the peripheral side the continuity of the tube is almost

or entirely lost from narrowing or actual obliteration. Sometimes the cavities communicate with one another.

The two forms of bronchiectasis often co-exist.

The walls of the dilatations undergo changes in the course of the disease. The mucous membrane becomes granular, swollen, and congested; while at a later stage it presents a velvety or villous appearance, and in some cases there is even ulceration with superficial necrosis. The muscular and elastic coats become atrophied, and coincidently with this dilatation increases. At times the wasting of these coats is partial; some portions of the walls retaining their natural volume, and forming bands or ridges elevated above the surrounding membrane. The dilated tubes occasionally present an appearance of hypertrophy; the walls are thickened, but the thickening depends on changes which have taken place in the mucous membrane. The cartilages resist the destructive metamorphoses longer than the other structures, but they sometimes partake of them. The contents of the tubes may be either muco-pus, or pus; and casts of the minute bronchi are met with. At times the contents are very fœtid. Crystals of margarin are occasionally found, and sometimes fragments of pulmonary tissue. It is said that the contents may become calcareous.

Dilatation of the bronchial tubes may be unattended with any change in the surrounding lung-tissue, but generally speaking, condensation of the latter takes place, either as the result of pressure or

of chronic pneumonia. In some instances the tissue forms an abscess, in the centre of which the walls of the bronchial tube are found, whilst in others the walls and the surrounding tissue are destroyed by gangrenous inflammation. It is generally not difficult to distinguish between a phthisical cavity and a dilated bronchial tube. The latter is not characterised by the broken irregular surface which usually exists in the former; its shape is generally more regular; and it is usually continuous with other tubes. The surrounding lung-tissue has no tubercular infiltration.

The *mechanism* of bronchiectasis has occupied much attention. It is probable that the elastic and muscular fibres lose their elasticity and contractility as the result of chronic inflammation, and thus yield to the distending influence of coughing. When once a dilatation is produced, accumulation of the secretions takes place, which tends further to increase the dilatation.

Symptoms.—The symptoms of bronchiectasis are those of chronic bronchitis aggravated in some important respects. The cough is frequent and paroxysmal. The expectoration is very abundant, very purulent, and when the disease has lasted some time, very fœtid. The breath also becomes fœtid. Hæmoptysis is occasionally met with, even to a considerable extent. There is more wasting than in ordinary bronchitis, and the blood is more imperfectly aërated. Night-sweats are not uncommon. In fact, the general symptoms approach those of

phthisis. The digestive functions are usually not much impaired.

Physical Signs.—The movement of expansion is diminished in bronchiectasis, while that of expiration is prolonged. Over the affected portions of the lung there may be slight retraction. Vocal fremitus is increased, and rhonchal fremitus is sometimes well marked. The percussion-note is altered. If a dilated tube is surrounded by condensed lung-tissue, or is full of secretion, there is dulness on percussion; but if it is situated near the surface and empty, some degree of tubular resonance may exist. Cracked-pot sound may be at times elicited. The respiratory sounds are harsh, or loudly bronchial with a more or less blowing character, and they may be distinctly cavernous. Vocal resonance is often greatly increased. The pulse becomes rapid in the later stages. The temperature rarely if ever reaches the height that it does in phthisis with cavities, and the daily oscillations are not so marked.

Diagnosis.—The main difficulty as regards diagnosis is in the differentiation of certain cases of phthisis with cavities, from bronchiectasis with large globular dilatations. The points to be relied on are that in the latter disease the morbid physical signs are usually met with at the middle and lower parts of the lungs, whilst in ordinary phthisis they are found at the apex; that the temperature differs in the two affections as mentioned above; that emaciation and night-sweats are not so marked in bronchiectasis; and that, if cases are watched, there is

usually observed a progressive advance of symptoms in phthisis, whilst in bronchiectasis the symptoms may remain stationary. In phthisis signs of consolidation precede those of cavities, whilst they follow them in bronchiectasis. Bronchial dilatations and tuberculous cavities have been found in the same lung. The fœtor of the breath and sputa in bronchiectasis may cause a suspicion that gangrene of the lung is present; but the general symptoms will usually enable the practitioner to differentiate between the two affections.

Prognosis.—Bronchial dilatation is probably never cured. It may last for years.

Treatment.—The treatment of bronchiectasis must be that of chronic bronchitis with the use of such measures as are applicable to wasting diseases in general. The fœtor of the breath is best relieved by the inhalation of creasote.

5. *Narrowing or Obstruction of the Bronchial Tubes.*

Narrowing or obstruction of the bronchial tubes is by no means uncommon, and may depend on intrinsic or extrinsic causes. Complete obliteration of a tube is sometimes found in connection with bronchiectasis, immediately beyond a globular dilatation.

Ætiology.—The *intrinsic* causes of obstruction are a thickening of the mucous membrane resulting from bronchitis; the retention of viscid secretions; the exudation of plastic material into the interior of the tubes; and the deposition therein of tubercle or cancer.

Amongst the principal *extrinsic* causes are: the pressure of adjacent tuberculous or cancerous deposits; the contraction of plastic matter exuded into the tissues surrounding the tubes; solid formations in the pleura; enlarged bronchial glands; and aneurismal and other thoracic tumours.

Obstruction is most frequently met with in the smaller tubes, but the pressure of thoracic tumours not unfrequently causes obstruction, or even obliteration of a main bronchus, which occasionally—as in the case of aneurisms—becomes perforated.

Symptoms.—If a large bronchial tube becomes suddenly and greatly obstructed, dyspnœa of an urgent character sets in, and death from apnœa may speedily result, unless the obstruction be removed. When the obstruction is on a smaller scale, being confined to the smaller tubes, or when a large tube suffers only from slow, gradually increasing obstruction, the symptoms are by no means urgent for a time, and slight dyspnœa, sometimes accompanied by stridor, is the most marked feature.

Physical Signs.—Complete obstruction of a bronchial tube invariably leads to collapse of the portions of the lung to which the tube is distributed, and thus an entire lung may collapse if its main bronchus be obliterated. Where partial collapse is produced, emphysema of the neighbouring lung-tissue commonly follows, and if one lung become collapsed, the opposite lung becomes enlarged and emphysematous. The existence of collapsed lung gives rise to dulness on percussion over the affected part, unless

this be situated away from the chest-walls, or masked by the presence of emphysema. Further, obstruction of the bronchial tubes causes a weakness or deficiency of the respiratory sounds, with a prolonged expiratory murmur, attended at times with sonorous and sibilant rhonchi. Over the collapsed portion of the lung, or over portions of the lung supplied by a tube which has become completely obstructed, the breath-sounds are absent. Deficiency or absence of vocal vibration is another physical sign of obstructed bronchial tubes.

Treatment.—The treatment of obstruction of the bronchial tubes must depend on the nature of its cause. The chief interest of the affection arises from the means of diagnosis of thoracic tumours which it may afford.

6. *Cancer of the Bronchial Tubes.*

Cancer of the bronchial tubes occurring independently of cancer in the lungs or mediastina, is probably never seen; but cancerous matter has been found in the tubes; (1), in cases where the lungs have been infiltrated with a similar deposit; (2), where a cancerous tumour connected with the root of a lung has perforated a tube; and (3), in some cases of cancerous disease of the lung; a tumour of a similar nature being found connected with the mucous membrane of a tube. (4), Cancerous matter has also been found *in transitu* in a tube, having been detached from a cancerous mass.

CHAPTER IV.

On certain Cases of Lung-disease resembling Acute Phthisis.

(CLINICAL LECTURE.)

Gentlemen,

There are certain cases of acute inflammatory disease of the lungs which in their clinical features so closely resemble acute phthisis, that the differential diagnosis of them is often exceedingly difficult; and I wish to-day to speak to you of such cases, and to refer to some which have come under my own observation.

We are familiar with two forms of acute phthisis; and let me draw your attention briefly to their chief characteristics before I allude to the special subject of my lecture.

First, we have that form of acute phthisis which begins with symptoms resembling those of pneumonia, with crepitation at one apex soon appearing in both apices, extending rapidly, and followed by consolidation and disintegration of the lung-tissue. Cavities soon form; there are copious expectoration, rapidly increasing weakness, profuse perspirations, quick pulse, and a temperature reaching a very high level in the evening, and sinking to the normal, or even sub-normal, in the morning. The debility is

great, but there is not the utter prostration, which is seen in the second form. Death usually takes place at the end of a few weeks, probably within three months from the onset of the attack; and, after death, we find the lungs more or less solidified throughout, with numerous cavities, but few or no tubercles. Such cases are generally met with in somewhat young patients who come of a phthisical family. To this form of the disease, we give the name *acute pneumonic phthisis*. It is a form of scrofulous pneumonia.

The chart (Fig. 10) which I show you will indicate to you the character of the temperature in a well-marked case of this kind; the temperature was taken between nine and ten in the morning, and between seven and nine in the evening. The patient, in this instance, was a young lady whom I attended some years ago. She did not live many weeks from the beginning of her symptoms. She came of a phthisical family, and, after her death, I attended her sister for a similar disease which ended fatally.

Secondly we have that form of acute phthisis, in which the symptoms at the beginning resemble very much those of typhoid fever; in which there is but little cough at first, with but little expectoration; in which the pulse is very quick, and the prostration extreme. In these cases the temperature is high, but it does not present those variations which characterise the first form. It maintains a more even level, rising slightly in the evening, but not

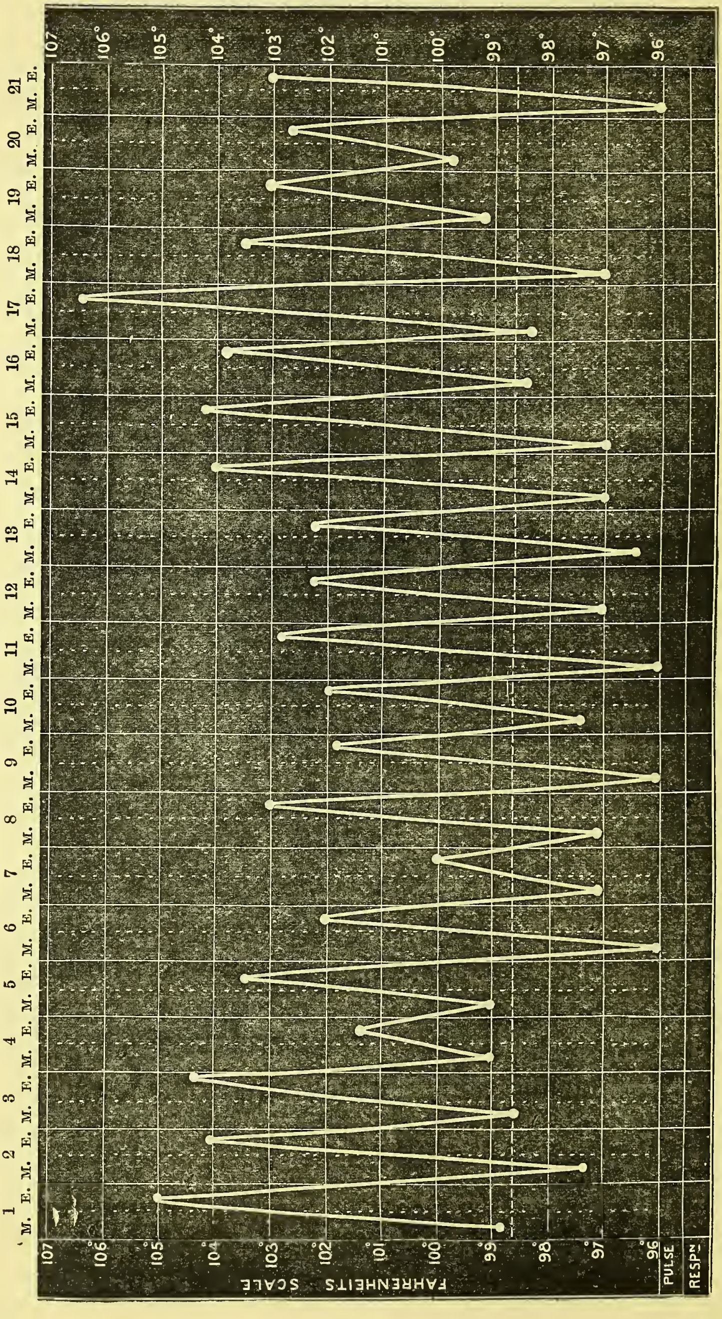
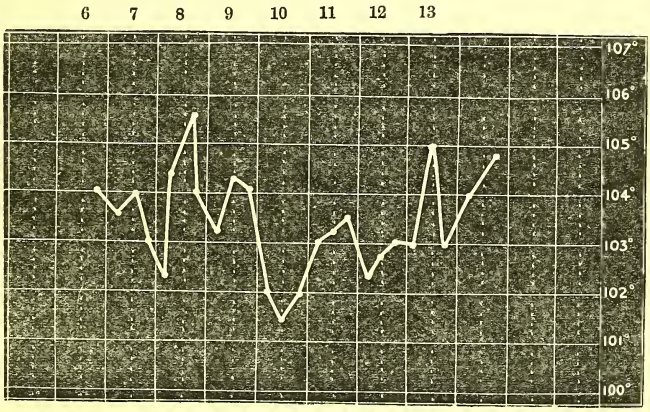


Fig. 10.—*June*, 1875.

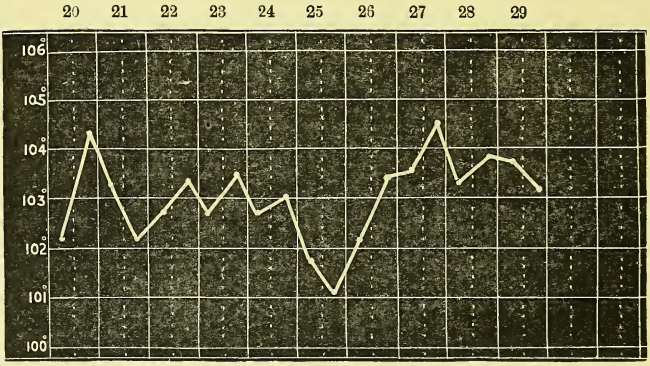
falling to the normal in the morning. The physical signs, though doubtful at first, soon show the nature of the case. We hear *râles* all over the chest,—fine subcrepitant and crepitant—indications of the existence of general capillary bronchitis, or pneumonia. These cases usually end fatally in a few weeks, and they are often accompanied by head-symptoms. We know that, when the patient dies, we shall find the lungs riddled with tubercles, around which the lung-tissue has become inflamed. To this form of disease we give the name *acute tubercular phthisis*. The subjoined charts (Figs. 11 and 12) show the temperature-ranges in two such cases.

Fig. 11.—*February.*



Now there may be, in such cases as those to which I have referred, little or no difficulty, except just at first, in forming a correct opinion as to their nature; but I have met with cases in which the clinical features so closely resembled those which I

Fig. 12.—*April.*



have described, that I have expressed a fear that they were really cases of acute phthisis, yet the patients have eventually recovered; and I have therefore been compelled to ask myself whether I had not made a mistake in diagnosis. Is it possible that cases of acute phthisis can recover?

Whilst I should unhesitatingly say that, when once the second form of acute phthisis has set in, and the lungs are studded with miliary tubercles, recovery is impossible, I think that, in the pneumonic form of the disease, recovery may take place. The affection is essentially a scrofulous pneumonia, and as such may be amenable to treatment. Again, just as we see that cases of subacute and chronic phthisis, especially those which begin in so-called catarrhal pneumonia—for I cannot, as the result of my clinical experience, admit that all cases of subacute or chronic phthisis begin with the deposition of tubercle—get well, so I think we may hope that cases of a more acute form may also recover.

Now let me draw your attention to the notes of the following case :—

Amy S., aged sixteen, was admitted into No. 15 ward on February 21st, 1877. It appeared that, up to twelve months before admission, she had enjoyed very good health. At that time, some dropsical symptoms appeared. Her abdomen began to swell. Under medical treatment, the swelling subsided in about five weeks, and she remained well up to three weeks before entering the Infirmary. On admission, she was suffering from ascites and œdema of the legs, unconnected, as far as could be ascertained, with any organic disease. Iodide of iron, and subsequently iodide of potassium, were administered, and the abdomen was painted with tincture of iodine. I considered the case as probably one of tubercular disease of the peritoneum, or else one of ordinary chronic peritonitis.

By February 27th, the œdema of the legs had disappeared, and the abdominal swelling was less, but a fresh symptom now set in. She caught cold, and a cough came on early in March ; and, on the 10th, she began to expectorate somewhat rust-coloured sputa. She was then ordered carbonate of ammonia and cascarilla, with four ounces of port wine daily.

On March 12th, the following were the physical signs elicited by careful examination of the chest. There was loud breathing, with moist sounds, at the left apex. There was some dulness, with crepitation and bronchial breathing, under the right clavicle, and dulness, but no crepitation or friction-sound at the right base. The breathing was rapid, and there was severe cough, especially on exertion. The pulse was quick, and the temperature high. I shall refer to these signs later on.

On the 16th, in consequence of the dulness over the right base having extended, and as I feared there might be some

fluid in the pleura, I punctured the chest, but none was found.

During the following week the symptoms increased in gravity. The dulness over the right apex extended downwards, the crepitation became coarser, and appeared to indicate the breaking up of the lung-tissue.

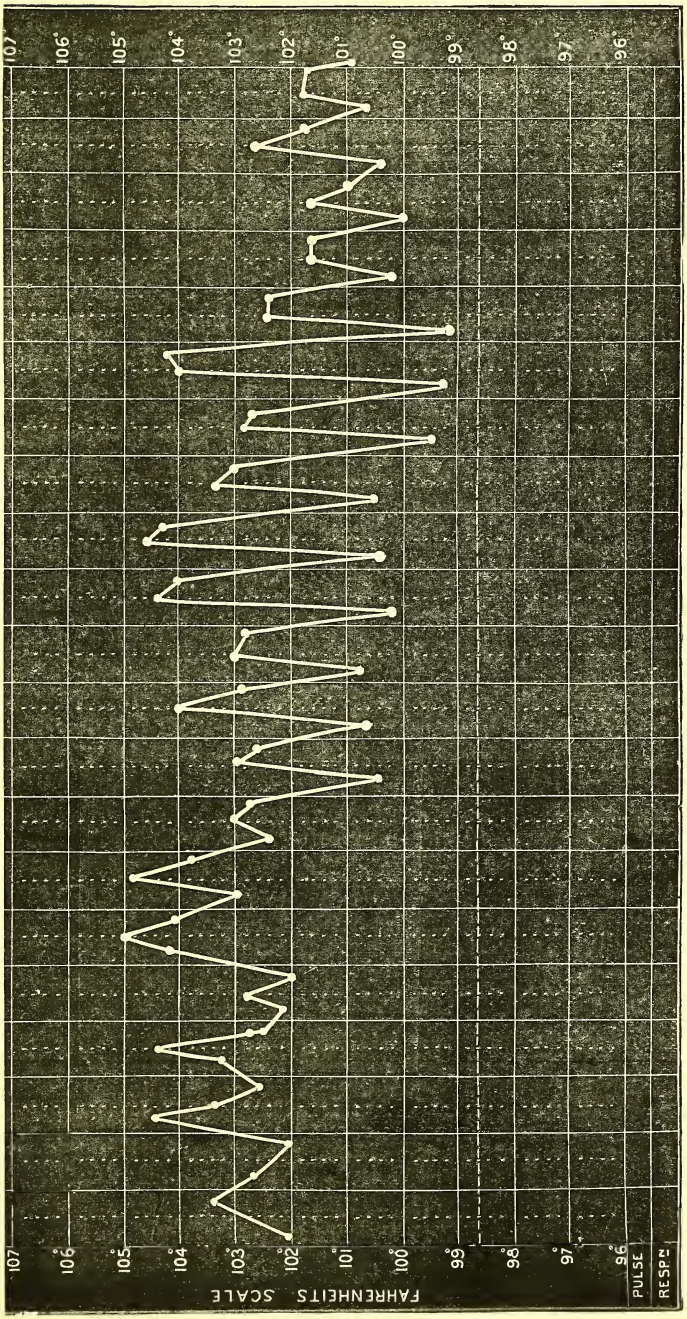
On March 29th, the fluid had almost entirely disappeared from the abdomen, and the general symptoms were somewhat less severe, but there was no improvement in the physical signs.

On April 7th, after the patient had been under treatment forty-five days, and about five weeks after the lung-symptoms had set in, there were dulness and cavernous (?) breathing under the right clavicle, with dulness and crepitation lower down, and coarse crepitation all over the back. Throughout the case, the mischief in the left lung was confined to the apex.

From about the middle of April, the patient began to improve, but it was not till near the end of June that she was able to be removed into the country.

Now, having referred to the physical signs—which were such as characterise acute pneumonic phthisis in its stage prior to the formation of numerous cavities—let me refer to the general symptoms. First of all, the pulse was very rapid; it varied from 140 to 120 in the earlier weeks, never falling below 110 till after the middle of April, namely, about seven weeks after the patient's admission.

And now with regard to the temperature; I wish particularly to call your attention to this. The charts (Figs. 13 and 14) show its variations. You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FAHRENHEIT'S SCALE
PULSE
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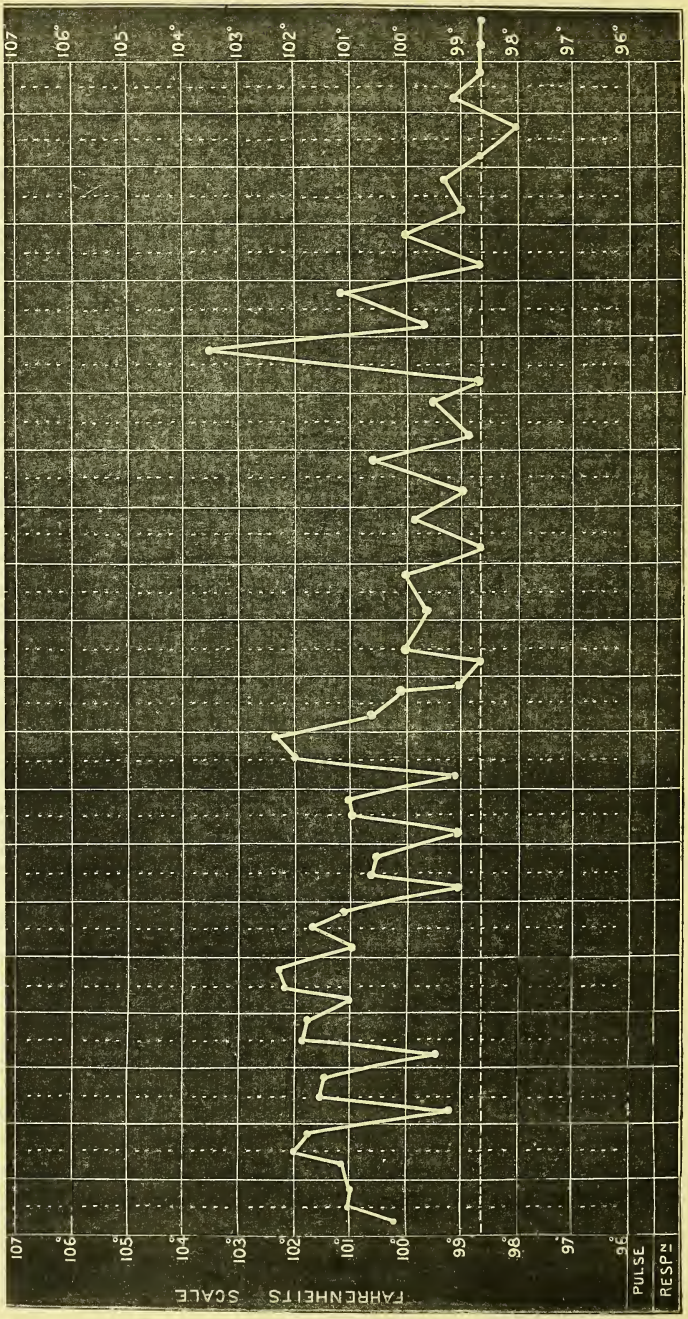
Fig. 13.—*March.*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107 106° 105 104° 103° 102° 101° 100° 99° 98° 97° 96°
FAHRENHEIT'S SCALE
PULSE
RESPN

Fig. 14.—*April.*

see that it maintained a high level for some weeks, but it had marked oscillations, being very high in the evening, and low, nearly normal, in the morning. You will observe that it was taken three times a day, namely, at 9 a.m., 5 p.m., and 9 p.m., and that it was generally found highest at 5 p.m. This, as you know, is a very frequent feature of the temperature in phthisis.

Thus we had in this case the characteristic high fever of acute phthisis; we had rapid consolidation of the apex of one lung, with indications of disease in the apex of the other lung; we had a steady extension of the disease, signs of the lung breaking up, and of the formation of a cavity, with great weakness of the patient. These symptoms lasted many weeks, and recovery was very slow.

On May 26th, the following physical signs were noted. There was still dulness over the right apex with blowing sound, as of a cavity; over the right back crepitation was still heard. The pulse was quiet, and the temperature normal. The patient was removed to the Convalescent Hospital at Woolton about June 20th, and the physical signs at that time were as follows. The respiration was almost natural at the base of the right lung; there was slight crepitation towards the middle of the lung. Breathing was somewhat bronchial at the upper part of the lung behind. In front, the breathing was loud and bronchial at the right apex, but less so at the left; otherwise, the sounds in the left lung were healthy. There was slight flattening at both apices, with deficient expansion.

The patient returned from the Convalescent Hospital on July 21st, greatly improved in health and general condition.

Her physical signs were as follows. There was some flattening under the right clavicle, with deficient expansion; resonance was somewhat impaired; expiration was prolonged; the breath-sounds were harsh; there was fair resonance over the back of both lungs, and the respiratory sounds were fairly good.

Now let me refer to the treatment that was adopted. When the chest-symptoms began, the patient was put on carbonate of ammonia and cascarilla, and for the latter cinchona was substituted on March 17th. On March 23rd, three grains of quinine in mixture were ordered to be taken three times a day; and, on the 26th, fifteen minims of tincture of digitalis were added to each dose. This treatment was continued until April 2nd, when iron and quinine were given. Subsequently, cod-liver oil was taken. From the date of her admission, and more especially when the lung-symptoms set in, nourishment was given very freely. It consisted of milk, beef-tea, and eggs, and solid food as soon as it could be borne; six ounces of port wine were also given daily from March 12th, and continued (except for three days from the 26th to the 29th, when four ounces of brandy were given) up to May 11th, when porter was ordered.

The case which I have thus presented to you is one of great interest. I think it belongs to the same class as that of which I showed you the temperature-chart at the beginning of the lecture; that it was truly a case of acute pneumonic phthisis,

or scrofulous pneumonia. It is very important to note the kind of treatment under which recovery took place, namely, free nutrition, and the administration of such remedies as tend to lower temperature and give strength.

I have had under my care a few other cases which have resembled acute phthisis, and which have recovered. I cannot give you to-day the details of all these, but I will refer briefly to two.

Margaret P., aged thirty-three, a servant, was admitted into the Infirmary under my care, on December 29th, 1880. She was suffering from symptoms resembling those of typhoid fever; but there was cough with extensive subcrepitant *râles* over both lungs. The pulse was very quick, and the temperature high. Her condition was such that I hesitated in giving a diagnosis; on the one hand I feared typhoid fever, and on the other acute tubercular phthisis. As the case progressed, the symptoms assumed more the character of the latter disease, and for some weeks the condition of the patient was very critical. We had evidence of extensive bronchitis, with some consolidation of the right lung. The pulse remained between 120 and 140. The temperature kept uniformly high, never, however, reaching a great height, and never falling to the normal. Its characteristics were those of the second form of acute phthisis, which I have referred to at the beginning of the lecture. The respirations were rapid, 40 to 52 in the minute, and the prostration was extreme. It was not till more than three weeks after her admission that the patient showed decided signs of improvement. From that date she steadily improved, and on April 17th she was sent to the Woolton Convalescent Hospital. I have not time to go into the details of her case, it must suffice to say that the treatment

adopted in it was similar to that which was used in the case of A. S., namely—nourishment given freely, stimulants and quinine.

The other case is the following:—

William C., aged fifteen years, a "coal boy," was admitted into the Royal Infirmary under my care on October 13th, 1886. The family history of the boy was bad, one brother having died of "water on the brain," and four or five others of bronchitis. (?) The boy had had no previous illness of importance, but about fifteen days before admission, without any previous sensation of being unwell, he was suddenly seized with severe and prolonged rigors, and had continued ill with pains in the head and abdomen, thirst, and fever, from that time. On admission there was an anxious expression of countenance, much prostration, a dusky face with a furred and glazed tongue, and anorexia. The abdomen was slightly tense and tender, the skin was hot and dry, and the temperature in the evening was 104 deg. Fahr. There was frequent severe cough with viscid muco-purulent expectoration, but no blood. The respirations were 40. There was slightly deficient resonance with crepitation at the right apex in front, and sonorous and sibilant rhonchi were heard all over the right lung anteriorly, and some crepitations and rhonchi were heard over the apex of the left lung. Behind, there was diminished resonance over the left apex and both bases, especially the right, but no tubular breathing. Some crepitations and rhonchi were heard all over the back of both lungs. The pulse was quick, but otherwise healthy. The urine was dark-coloured, free from bile and albumen, and there was no diminution of chlorides. From the general appearance of the boy, from the fact that the illness had existed for a fortnight, that but little consolidation of the lungs had taken place, the case was clearly not one of

ordinary acute pneumonia. I feared that the disease might be acute tuberculosis in its early stage. As the case progressed, the temperature assumed the character which is common in this latter affection. It was taken three times a day, viz., at 9 a.m., 5 p.m., and 9 p.m. It was generally highest at 5 p.m., and in the morning it was comparatively low. However, I expressed an opinion that the case might possibly be one similar to those which I had previously seen, and of which I have given you details. The treatment I adopted was to put the boy on as good diet as he could take; nourishment—strong beef-tea and milk—was given frequently, with brandy, and he was ordered the following mixture :—℞ Sod. Hypophosph. gr. xx ; Tinct. Quininæ ʒvj ; Syrupi ʒij ; Aq. ad ℥vj ; ℥j. t. d. s.

For the first few days there was but little change in the symptoms ; the pulse kept quick, the temperature-oscillations were very marked, and the crepitations became rather more extensively heard over the lungs, but the extent of dulness did not increase. Examination of the sputa on the 16th of October did not reveal the presence of any tubercular bacilli. On the 18th, the boy brought up some chalky-looking pellets in the expectoration. About the 21st, a decided remission of the symptoms occurred, the temperature fell, and in a few days reached the normal. A steady improvement now set in, and the physical signs began to improve. He gained strength slowly, and it was not till nearly the end of November that the physical signs had resumed the normal character. He was sent to the Convalescent Hospital, at Woolton, apparently quite well, on December 2nd. The treatment by free nutrition, quinine, and hypophosphite of soda, was continued throughout, and cod-liver oil was given from the 25th of October.

These cases which I have now referred to may serve as illustrations of the class of cases of which

we meet with occasional instances. In the early stages of the affection you must be very guarded in your prognosis. The most important points in regard to treatment, in my opinion, are that your patients should be put to bed as early as possible, that they should be carefully nursed, that free nutrition should be administered with a moderate amount of alcohol, and that such medicines as I have referred to should be given; the greatest care being taken not to disturb the digestion in any way.

CHAPTER V.

On Cancer of the Lungs.

(CLINICAL LECTURE.)

Gentlemen,

I purpose occupying the time at our disposal to-day in considering, very briefly, the subject of primary malignant disease of the lungs. There is probably no affection of the chest in which more mistakes of diagnosis are made than in this disease; and undoubtedly it is often very obscure in its origin, and very insidious in its course. In a large number of cases, accurate diagnosis in the early stages is almost impossible; but yet I think that, as a rule, by carefully considering the features of any given case, its mode of development, and the local and general symptoms which characterise it, we shall, even when the disease is not very far advanced, be able to arrive at a well founded conclusion as to its nature.

Cancer of the lungs usually comes on in a very insidious manner, and in its earlier stages may and does simulate, and indeed frequently is complicated with, pleurisy, pleuropneumonia, or bronchitis. One of the first symptoms which calls attention to the disease is often a slight pleuritic attack, or pain resembling that of pleurisy or pleurodynia. On

making an examination of the chest, you may find a slight dulness at the base of one lung, and possibly a friction-sound. You think you have to deal with a simple case of pleuritic effusion, and you use the measures which you consider appropriate to such affection. But the symptoms do not yield; the dulness over the lung slowly extends, the patient begins to emaciate, he becomes weaker, his pulse rises, but the increase of temperature is slight, only to the extent of two or three degrees.

Again, instead of finding a friction-sound when you examine the chest, you may hear crepitation or bronchitic râles, and you think the case is one of subacute pneumonia or bronchitis. There may even be expectoration of rust-coloured sputa; but for the most part, in cancer, the sputa are whitish, tenacious, very difficult to expectorate, and occasionally bloody. There is often a cough of a very severe kind, and paroxysmal in character, which does not yield to ordinary remedies.

As the disease progresses, and you find that the constitutional measures to which you have resorted for the supposed pleuritic effusion or pneumonia have not the desired effect, you begin to doubt the correctness of your diagnosis; and with the view of satisfying yourself on this point, or possibly thinking that some fluid, serous or purulent, exists in the pleural cavity, which should be removed, you make an exploratory puncture into the chest, either with an ordinary hypodermic syringe, or with a syringe such as I use in such cases; or you at once

use the aspirator, and you find that no fluid is withdrawn, or, perhaps, only a few drops of blood. You have, in fact, punctured a solid lung, and, in a measure, you have cleared up the diagnosis.

Now, you must not imagine that, in making a puncture of this kind, although you have performed an useless operation, you have done anything which seriously injures the lung or jeopardises the patient. If only the hypodermic syringe, or a fine trocar and cannula be used, no harm is done. It is unfortunate not to find fluid, if you have expressed a strong opinion to the patient or the patient's friends that you expected to find it; but, if you have been guarded in the opinion you have expressed, and you certainly should be so, your mistake or failure will not seriously compromise you. Such mistakes, indeed, cannot be avoided. The differential diagnosis between a cancerous lung or a lung solid from some other cause, and pleuritic effusion, is at times so difficult that the most accomplished and most experienced physician may make a mistake with reference to it; and in all cases of doubt an exploratory puncture should be made.

There is, in addition to pleuritic effusion and consolidated lung, another condition which may simulate cancer of the lung. I mean diffused thoracic aneurism. It is occasionally difficult in a case—when we have even eliminated the question of pleuritic effusion — to say whether a dulness which we find in the chest is due to an aneurism which has become diffused, or to a cancerous lung.

Some years ago, I attended a lady who developed symptoms of pleurisy in the left side; and soon after the onset of the symptoms there was distinct pulsation in the upper part of the chest, which I at first thought to be due to an aneurism. As the case progressed, however, the symptoms became less equivocal, and the patient died with all the indications of malignant disease of the lungs.

Again, I was called, some years ago, to a gentleman in Cheshire, who, after having had a fall, developed symptoms of pleurisy—pain in the side, etc. When I saw him, there was dulness over the lower part of one lung behind, and I expressed the opinion that the case was one of malignant disease; but I did not feel quite sure of my diagnosis, for the conditions which I met with might have been due to an aneurism produced by, or existing at the time of, the fall, and subsequently becoming diffused. The gentleman died not very long after I saw him; but no post-mortem examination was allowed.

Cancer of the lungs often exists for a long time, and may go on to a fatal issue without causing much, or indeed any, pain. There may be no marked cancerous aspect, but the gradual emaciation and increasing debility show the serious character of the disease. Dyspnœa, sometimes only slight, but occasionally severe and paroxysmal, is usually complained of; but in many cases this difficulty of breathing does not trouble the patient much, and it may be difficult to persuade him that there is any-

thing seriously wrong with his lungs, so slight are the subjective symptoms as regards these organs.

Let me refer now a little more in detail to the subject of diagnosis. The great difficulty, for the most part, is in distinguishing between malignant disease in the early stages, and pleurisy; and the points of difference are these. In cancer, the dulness, although it may be very decided, simulating that of fluid, does not vary in site with change of position of the patient; it does not, as a rule, even when the whole lung is involved, extend beyond the median line in front, as is the case in extensive pleuritic effusion. Sometimes, however, a cancerous development in the anterior mediastinum does produce a dulness which passes the median line, and even a solid lung may slightly encroach on the opposite side. The heart is not usually displaced to any material extent. There may, however, be but little displacement of it in pleuritic effusion in consequence of its having contracted adhesions. In extensive pleuritic effusion, when the chest is quite full, the dulness is complete in all positions of the patient, but in such cases there is usually cardiac displacement.

But in many cases of malignant disease of the lungs, there is some pleuritic effusion; and the fact that on tapping you draw off fluid may mislead you as to the real nature of the case with which you have to deal. There was a little girl in No. 15 ward some years ago, who was admitted with the following symptoms. There was well marked dulness over

nearly the whole of the back of one lung, with some crepitation at the upper part. I thought there was fluid in the pleura, and I tapped the chest, and drew off about fourteen ounces of serum. But the symptoms did not yield; the girl grew gradually worse, and I tapped her a second time, but only a small quantity of bloody fluid was removed. She died, and we found the lung a mass of cancer.

In the progress of a case of cancer of the lungs, symptoms of pressure often show themselves. There may be some dysphagia from pressure on the œsophagus; and one or both arms, and the neck and face, may begin to swell from pressure on one or both of the innominate veins, or the superior vena cava. In one case which was under my care in the Northern Hospital, the œdema was most marked. The patient's arms, face, neck, and thoracic trunk became enormously swollen, whilst the lower limbs and the lower part of the trunk were free from swelling. The appearance of this man was very peculiar. Another symptom which occasionally shows itself, and which helps in the diagnosis, is an enlargement of the lymphatic glands about the root of the neck from cancerous infiltration.

It sometimes happens that cancerous affection of the lungs is confined to one side of the chest, one lung becoming quite a mass of cancerous disease, whilst the other remains free from it. In the case to which I shall shortly refer, one side only was affected.

In the development of the symptoms and signs of

cancer, much depends on the site in which the disease originates. If it begin in connection with the pleura, the symptoms of pleurisy predominate; if, on the other hand, it originate in the interior of the lung, the disease may simulate bronchitis or pneumonia, or some other form of consolidation of the lung. There are cases which, in their physical signs, very closely simulate phthisis; but the temperature-ranges in them are not like those of the latter disease. The temperature maintains a much more even level, and rarely rises to any great height. The character of the breath-sounds will not help you much in your diagnosis of an obscure case of cancer. They may be altogether absent, as in extensive pleuritic effusion, or in a lung intensely solidified from some non-malignant disease; or they may be feeble. For the most part, there is a certain amount of movement of the affected side; vocal vibration is usually present, but is not strongly marked.

In regard to treatment, I need not tell you that cases of cancer of the lung are hopelessly incurable. We know of nothing which will beneficially influence the course of the disease. You must treat symptoms as they arise. If the case be complicated with pleurisy, with or without effusion, you may often give relief by such measures as you would adopt in ordinary pleurisy—poultices, etc., or tapping. For the most part, your attention must be directed to relieving pain and distress, often very prominent features, and to giving such food as the patient can

take. The hypodermic injection of morphia, or the administration of opium in some form, is often of great service; it relieves pain, soothes the patient, and gives him sleep. Cancer of the lungs is sometimes rather slow in its progress, and the aid that you can afford will tend to minimise the sufferings which are more or less incidental to the disease.

And now, having made these remarks, let me draw your attention to the following case.

James C., aged forty-four, was admitted into the Infirmary under my care on January 13th, 1886. He complained of tightness in the chest and slight dyspnœa, but of no pain. His illness began, he said, about three months before his admission, and he had been gradually wasting since then. He had, however, followed his work in an iron foundry up to six weeks before coming to us. On examination, we found the whole of the right side of the chest dull on percussion, with very impaired movement. The dulness was very marked, but it did not extend beyond the median line in front. Vocal fremitus was very slight. The breath-sounds were very faint all over the lung. On the left side, the resonance was good, and the breath-sounds loud, but healthy. The heart was not displaced, and the sounds were normal. The aspect of the patient, although somewhat pale and sallow, was not otherwise unhealthy. The pulse was 85. On the day after admission, the right pleura was tapped, and twenty ounces of a dark-coloured fluid were drawn off by the aspirator. The fluid was very fibrinous, and coagulated soon after it was drawn from the chest; indeed, it coagulated in the cannula during the operation. There was a slight alteration in the physical signs after the tapping; the breath-sounds were more audible, but the patient did not improve. On January 17th, tapping was again practised,

but only two ounces of fluid were withdrawn. The man now became rapidly weaker, and took his food badly. He complained of restlessness and insomnia, but there was no pain. The physical signs in the lungs remained unchanged. There was some expectoration of frothy, tenacious, and rust-coloured sputa, for a few days rather abundant. The symptoms of exhaustion gradually increased. The chest was punctured again on the 25th, but only a very small quantity of blood-stained fluid was withdrawn. About February 1st, the abdomen began to swell, but there was little or no pain. During the whole time the patient was under observation, his motions were very pale, but there was no jaundice. The temperature remained moderate throughout, never rising above 100 deg. The pulse became quicker as the debility increased. He died on February 7th.

I expressed an opinion when I first examined the man, that he was suffering from malignant disease of the lung, and the results of the tappings tended to confirm the opinion. At the same time, the case was just one of those in which the differential diagnosis between pleuritic effusion and cancer is very difficult, and the disease was complicated, as it occasionally is, with pleuritic effusion, thus making the diagnosis less easy. From the results of the post-mortem appearances, I think there can be no doubt that the affection commenced in the pleura, which became infiltrated with malignant deposit, much thickened and inflamed, that extension to the lung took place subsequently, and that the further spread of the disease to the abdominal cavity occurred during the last few weeks of life.

I need say but little about the treatment. In addition to the tapping, which gave relief, I ordered carbonate of ammonia, and subsequently quinine; and sedatives were used to procure sleep.

The following are the notes of the necropsy, furnished by Dr. Barron.

The left lung and pleura were healthy; the right pleura was occupied by a diffuse malignant growth. Two spaces still existing in the pleural cavity contained yellowish glairy fluid. The right lung was infiltrated with malignant growth, running along the course of the fibrous septa from the pleura into the lung-substance. The infiltration of the lung was most marked along the diaphragmatic and lower costal aspect. The lung was somewhat softened and breaking down at the base. The right arch of the diaphragm was infiltrated with the growth. There was some pericarditis, with malignant deposit on the parietal portion of the pericardium. The small and large omenta and the peritoneal ligaments of the liver were infiltrated with the malignant growth; there was recent diffuse peritonitis, with fluid in the abdomen. The liver was healthy. The cancer was of the carcinomatous kind.

CHAPTER VI.

Pleuritic Effusion.

(CLINICAL LECTURE.)

Gentlemen,

I wish to call your attention to-day to some cases of pleuritic effusion which have been in my wards, and to make them the subject of some remarks on the affection. I shall confine myself for the most part to those points which I consider of the greatest practical importance, and first I will refer to the subject of diagnosis.

It may, perhaps, appear to you to be very easy to recognise the existence of pleuritic effusion—to differentiate between liquid and solid matter within the cavity of the chest—and yet it is in some cases by no means so. I have known physicians of great practical experience mistake a solid lung for pleuritic effusion, and pleuritic effusion for a solid lung, and I have not been myself altogether free from such errors. There is, indeed, no single sign which invariably exists, by the recognition of which the diagnosis of pleuritic effusion can in all cases be certainly established, and it is undoubtedly true that the characteristics of its presence on which we mainly rely may, and often do, lead us into error.

Let me say a few words in reference to the evidence we derive of the existence of effusion from *percussion*. It is obvious that any solid or liquid in the chest will give rise to dulness on percussion; and it can only be by the character of the dulness, or its shifting nature, that we can say that it depends in any case on pleuritic effusion. Undoubtedly there is usually—indeed in the great majority of cases—a profound character about the dulness which can scarcely be mistaken; but there are exceptional cases of extreme dulness without any effusion. You may recollect the case of B——, in No. 10 ward, who came to us with a history of pleurisy, in whom we found dulness of a very leaden character, over the whole of the left side, extending up to the clavicle, and passing to the extreme right of the sternum. The breath-sounds were absent below, and only heard faintly at the upper part of the chest; moreover there was absence of vocal vibration, and the heart-sounds were faint and best heard to the right of the sternum. The presumption that pleuritic effusion existed was very strong, and it was thought desirable to ascertain the fact, so that, if fluid were present, some of it might be drawn off, and thus the urgency of the symptoms be relieved. A fine cannula was accordingly introduced, and the aspirator was used, with the result, however, of drawing off only a few drops of blood. Not satisfied with one exploration, I subsequently repunctured the chest at a different spot, but the result was the same.

The progress of the case showed its nature; faint crepitation was heard after a time, and death revealed the actual condition of the lung. It was more or less solidified throughout, and universally adherent to the chest-wall; the pleuræ were greatly thickened; and there were strumous deposits in the anterior mediastinum. These deposits had caused the dulness, which extended to the right margin of the sternum, producing thus a sign which, taken with the other signs, I had never previously met with in any lung disease except pleuritic effusion and cancer.

Again, in reference to the shifting nature of the dulness, you must not, in making a diagnosis of pleuritic effusion, depend too much on the fact, which I have often demonstrated in the wards, that the line of dulness varies according to the position of the patient. If the lung is perfectly free from adhesions, the fluid in the chest will gravitate to its lowest part, and the upper line of dulness will vary according as the patient is sitting or lying; but some of you will recollect the case of the woman in No. 15 ward in whom we had marked dulness, with absence of breath-sounds, in front of the left lung reaching to the level of the second rib, with resonance at the back extending even below the angle of the scapula, and from whom we drew off, at the time these signs were present, a large quantity of pus.

Auscultation often affords valuable aid in the diagnosis of pleuritic effusion. Speaking generally,

the breath-sounds are usually either absent or faint over the seat of effusion; but they may be also absent over an intensely solidified lung, or over one which is less solidified but adherent by very dense pleuræ to the chest-walls, just as was the case in B——, to whom I have referred.

Again, the breath-sounds may be very loud, simulating those of a solidified lung, when there is a large pleuritic effusion. There was a woman under the care of my colleague, Dr. Glynn, some time ago, in whom loud bronchial breathing was heard both over the front and back of the right lung, where there was marked dulness, and yet, as was subsequently proved, a very large quantity of fluid existed in the pleural cavity. In children, again, the phenomena of bronchial breathing and bronchophony are often present, although the effusion may be great; and I have met with other instances, besides the one I have alluded to, where loud breathing has been heard in adults. Moreover, you must not forget that in old-standing cases of effusion the sound lung takes on increased action, the breath-sounds produced in it become puerile, and may sometimes be heard on the opposite side of the chest.

But to take another sign, to which great importance is very properly attached. In pleuritic effusion it is undoubtedly true that *vocal vibration* is generally absent; that when the hand is placed on the chest whilst the patient speaks, no thrill is communicated to it; and yet I have sometimes felt a well-marked

vibration over a chest from which I have immediately afterwards removed a large quantity of fluid. Some of you may recollect the case of A——, in No. 10 ward, who was the subject of empyema. In that case I pointed out to the class that we had most of the signs of pleuritic effusion well-marked—viz., leaden dulness, absence of breath-sounds, etc. The man had been previously tapped, and a considerable quantity of fluid had been withdrawn. We had watched the gradual re-accumulation of the fluid, and the time had come when I resolved to tap again. Over the affected side—over the seat of leaden dulness, and where the breath-sounds could not be heard—there was distinct well-marked vocal fremitus. An aspirator tube was introduced, and we drew off ninety ounces of pus.

I removed, some time ago, two pints of serous fluid from the chest of a man in whom vocal vibration was distinctly perceptible, except at the extreme base of the lung—perceptible where there was marked dulness from the presence of fluid. Again, there was the case of the woman McK——, in No. 15 ward, who was admitted with pleuritic effusion, and was tapped several times. Before the first tapping you may recollect that there was distinct vocal vibration at the lower and back part of the affected side of the chest. I removed twenty-five ounces of fluid. Strange to say, we never had any return of the vibration throughout the progress of the case, although fluid collected and re-collected, and we removed it on three or four occasions.

I think that, possibly, we may account for the persistence of vocal fremitus in some of these cases of pleuritic effusion by the existence of adhesions of the lung to some parts of the chest-wall. But in other cases post-mortem examination has shown that no adhesions existed.

Wherever pleuritic effusion is great there is *displacement of viscera.* The heart is often found beating to the right of the sternum when the effusion is on the left side, or it is pushed towards the left axilla if the effusion is on the right side, and this displacement is greatest when the effusion has been rapid. There are also displacements from the diaphragm being pushed down. But, independently of the fact that displacements may be due to the presence of solid matter in the chest, it frequently happens that although there is a great deal of fluid in the pleura, there is but little visceral displacement. In the first place the heart may be so connected with the chest-wall by adhesion that it cannot be displaced except to a slight extent; and further, in the more chronic cases, the lung yields to the pressure of the fluid—collapses in fact—and thus leaves a large space for the fluid. The displacement of viscera may, therefore, be much less than you would expect from the quantity of fluid which, after operation, you find has existed.

Again, you must not always expect to meet with a decided increase in *the size of the affected side,* or a *bulging of the intercostal spaces.* Setting aside the fact that measurements are not always trustworthy,

it is undoubtedly true that in adults effusion may be very great and yet there may be no increase, as shown by the tape, in the size of the affected side, as compared with the opposite one. In the more yielding chests of children it is otherwise, and a notable increase is more frequently met with. Doubtless in most cases, if you were to watch them from the beginning, having taken the measurements before effusion, you would find some increase in the size of the affected side. As an instance of what I have just said, there is a case which you have seen in No. 10 ward. In this man, who was the subject of extensive empyema of the left side, the measurements before tapping were as follows:—Right side, 17¾ inches; left side, 17 inches. We drew off fifty-eight ounces of pus from the left pleural cavity. Now, it is very probable that the above measurement of the left side was greater than it would have been in health before the effusion took place; but the left lung being crippled, the right had taken on increased action, and had distended that side of the chest beyond the normal.

Again, although the intercostal spaces are at times altered in their appearance, becoming more or less convex, yet extensive effusion may exist in adults without any such change taking place.

It is scarcely necessary for me to allude to *ægophony* as a sign of pleuritic effusion. I look upon it as a mere fancy sign, being generally absent when there is any difficulty of diagnosis.

I have thus endeavoured to deal with some of the difficulties which you may meet with in the diagnosis of pleuritic effusion, and whilst I admit that in the majority of cases the diagnosis is easy, I must confess that in others it is very difficult; indeed, I think in some instances it is impossible to say positively whether fluid is present without making an exploratory puncture; and in all cases of doubt, where the propriety of tapping the chest is in question, no decided opinion should be pronounced until an exploratory puncture has been made. But I must say a few words about this exploratory puncture. Simple as it may appear, easily as it is accomplished, and usually attended in hospital practice with but little trouble, it is far less simple amongst private patients. It may become in fact magnified into an "operation," and should no fluid be withdrawn the confidence of the patient in the physician is not increased. Therefore, it is well to weigh carefully every feature of a case before introducing even the finest cannula. I believe, however, that no harm is done by the use of these tubes, even if a solid lung, or solid tumour, or a healthy viscus is punctured. You need not, therefore, be under any apprehension on that score. But I must tell you that even when there is a good deal of fluid in the pleura, one, or even two, punctures may fail to withdraw any of it. You may possibly puncture at a spot where there are adhesions; and, again, you may find that, even when there is a pure serous effusion, such as you would

think ought to flow through a fine cannula, nothing will follow the introduction of the tube, unless the exhausting syringe be used. I had under my care a man who, having had empyema of the right side, had symptoms of pleurisy on the left. On examination I recognised, as I thought, the presence of effusion. I introduced a fine aspirator-cannula, and I felt that I had passed the instrument into a cavity, but no fluid oozed out. I withdrew the cannula to see if it were plugged, but it was free. I introduced it again, but still no fluid appeared. The aspirator was applied, and twenty ounces of clear serum were drawn off. Sometimes from the extreme thickness of the thoracic-wall it is necessary to pass the cannula very deeply before you feel that you have reached the cavity of the pleura. You may recollect the case of the man N——, who was under my care in No. 10 ward. He had been previously tapped for empyema, and he had the signs of a re-accumulation of fluid. I introduced a cannula into the back of the chest. I felt the instrument entering a soft substance after having passed some distance through the chest-wall. I moved the cannula about, but clearly it was not in the pleural cavity. Had I made a mistake? Had I punctured a solid lung? The cannula was already buried two inches in the chest-wall. I pushed it further in, and had the satisfaction of finding that it entered a cavity. I drew off forty-five ounces of thick pus.

In this case, as in others which I have met

with, I have no doubt that the instrument which was introduced, after passing through the chest-wall much increased in thickness from deposit, pushed in front of it some soft pleural membrane, before perforating it; and an occurrence of this kind should warn you never to use a blunt instrument either in exploring or tapping the chest.

When you puncture the chest, if nothing but blood or bloody matter exude, it is well to examine the contents of the cannula under the microscope, for such an examination may enable you to differentiate between a cancerous and some other tumour. At the same time, it must be borne in mind that a cancerous lung may be punctured half-a-dozen times without a cancerous portion being reached, and cancer may exist although only blood is drawn off.

What I have said as to the non-passage of fluid through a cannula will teach you not to trust to the mere introduction of a grooved-needle as a means of diagnosis. What is largely used now is the ordinary hypodermic syringe, and, as a rule, it answers well; but sometimes, in cases of empyema, the pus is so thick that it will not pass through the fine tube of such a syringe. The instrument which I use is one made expressly for the purpose. It is like a hypodermic syringe, but somewhat larger, and with larger cannulæ. Occasionally this fails to withdraw the fluid, which will, however, appear if the pneumatic aspirator is used. In introducing the instrument, whatever you use, it is well to give it a semi-rotatory movement.

I must now pass on to say a few words about the treatment of pleuritic effusions, and my remarks will be confined chiefly to those which are more or less chronic. As a matter of fact, acute primary pleurisy is a rare disease. Pleuritic inflammation is usually connected with some constitutional state—some constitutional vice, and is more or less secondary. In reference to the more chronic cases, the treatment I have found most useful in promoting absorption of the fluid is the internal administration of Iodide of Potassium, with tonics, bark, etc. Iodide of Iron or the Tincture of the Perchloride of Iron may sometimes be given advantageously, and counter-irritation should be used. I prefer for this the application of Iodine rather than blistering, but blistering is often useful. I recommend you, however, not to blister severely. I have seen very large effusions which had lasted many weeks, even months, removed by this kind of treatment; and it is well, unless certain urgent symptoms are present, to give constitutional measures a fair trial before resorting to tapping. I must tell you that I have no faith in the powers of Mercury to produce absorption of these effusions, and I am not in the habit of prescribing it. I think it is important to keep up the strength of the patients, for there is a far greater probability of the fluid being absorbed when they are strong than when they are weak, and, therefore, good diet should be given with tonics, and in some

cases wine, whilst the special remedies are administered.

I will defer the consideration of the treatment of pleuritic effusions by tapping to another lecture.

CHAPTER VII.

Pleuritic Effusions—Their Treatment by Tapping.

(*CLINICAL LECTURE.*)

Gentlemen,

In a former lecture I spoke to you of the symptoms and physical signs of pleuritic effusion, and I made a few remarks on its constitutional treatment. To-day I wish to call your attention specially to the subject of *paracentesis thoracis* in the affection, and I shall endeavour to make my remarks as short and practical as possible.

The operation of tapping the chest, which used to be looked upon as a sort of last resource in cases of pleuritic effusion, has of late years become one very frequently witnessed in our wards, and there can be no doubt that by means of it many cases of pleurisy which would formerly have terminated fatally have been conducted to a successful issue. In this hospital, and indeed in all the hospitals of this part of England, pleuritic effusion is quite a common malady, and many cases pass under our notice every year. However, I do not wish to say anything as to the general nature of the disease, or the causes which render it so prevalent here, but

to confine myself strictly to the question of its treatment by tapping.

First, let us consider the circumstances under which pleuritic effusions occur.

Pleuritic effusions occur under various circumstances, and they differ very much in their nature. We may, however, classify them under the following heads :—

1. Effusions arising as the result of acute inflammation of the pleura.

2. Effusions due to chronic inflammation of the pleura, and

3. Effusions resulting from passive exudation, as in heart disease, Bright's disease, or emphysema of the lungs.

The fluid which is poured out into the pleural cavity varies much in character, according to the nature of the exciting cause; it may be serous, fibrinous, sanguineous, sero-purulent, or purulent.

Setting aside all question of constitutional treatment by medicines, counter-irritation, diet, etc., the point to which I wish to call your attention is the propriety of tapping in the disease, and the circumstances under which the operation should be performed; and in considering this, I think we cannot do better than take up the matter in connection with the three forms of effusion, viz., acute, chronic, and passive, in the order in which I have placed them.

First then, as regards those effusions which result from acute inflammation. Is it ever desirable to tap

in such cases? And if so, what are the conditions which call for the operation? To the first question, I answer emphatically in the affirmative. Although most cases of this kind, especially when the fluid poured out is small in quantity, will do well under constitutional treatment, and the patient will ultimately recover, yet there are some cases where, in my opinion, tapping is imperatively called for, and there are others in which the operation is desirable in order to save time, and promote a speedy recovery. The cases of acute effusion in which tapping is imperatively called for are those in which the effusion is poured out rapidly in large quantities, where there is much displacement of viscera, and, especially if there have been anything like urgent dyspnœa or threatened syncope; and should the patient who is suffering from these symptoms be either old or debilitated the more necessary does the operation become. Under such circumstances tapping should be resorted to without delay, and, probably, most physicians have known of cases where death has resulted apparently from the neglect to perform the operation. But, further, in cases of acute effusion, either the result of a simple pleurisy or a pleuro-pneumonia, even when the quantity of fluid in the chest is not excessive, it is, in my opinion, desirable to tap if, after a moderate use of constitutional measures, no improvement should take place, and if pyrexia should continue, and especially if the latter should assume a hectic type. I have now performed the operation

many times in cases of acute disease, and I have never seen any other than good results; in fact, I may say that, although I have seen cases in which I have regretted that the operation had not been previously performed, I have never had occasion to regret any early operation which I have resorted to.

The time at which the operation should be performed must depend on the circumstances of each case. It may be required at the end of ten days or a fortnight, or more, from the commencement of the attack; but delay is often not unattended with danger.

In illustration of my views I will refer to the following notes which will serve to indicate the kind of case in which the operation is desirable.

I attended a few years ago a gentleman who, having had slight hæmoptysis the previous summer, was attacked, after exposure to severe cold one night, with left pleuro-pneumonia. At the end of a week there were decided indications of pleuritic effusion, with much consolidation of the lung. The fever was high. No diminution in the quantity of fluid resulted from the measures which were resorted to, and at the end of a fortnight, viz., on the thirteenth day of my attendance, the patient was tapped, and forty ounces of clear serum were drawn off. The operation was stopped before all the fluid was withdrawn. No re-accumulation of fluid took place. The patient, who had a strumous aspect, and in whom a high temperature—a temperature such as characterises tuberculous cases—existed for some time, steadily recovered.

The removal of a portion of the fluid in this

instance at an early period tended, I believe, in large measure to promote recovery. It favoured the absorption of the fluid that remained, and gave room to the lung to expand, and thus prevented that sad result of neglected pleuritic effusion, a carnified or collapsed, or in other words, a more or less crippled or even useless lung.

And this leads me to say a few words on this latter subject. Whenever a pleurisy takes place the lung becomes more or less covered with a layer of lymph, which subsequently becomes organised and contracts, thus constricting the lung-tissue. If fluid effusion is present at the same time, the lung is pressed upon by it, and retracts towards its root. It thus becomes gradually smaller and in a worse condition for expansion, so that each day's delay in the removal of the fluid diminishes the chances of its recovering its normal size, and therefore its normal function. We are all familiar with the fact that when pleuritic effusion becomes absorbed there is often a great falling in of the chest-walls. The lung is unable to expand as the fluid diminishes, and great deformity and impairment of lung power is the result. And it must be borne in mind, that when the effusion is most inflammatory this condition of things is most likely to occur; whereas when the effusion is the result of passive exudation, and no lymph is deposited on the surface of the lung, constriction does not take place. The lung may collapse from pressure, but it will resume its original size if the pressure is removed. Hence the

great importance of not delaying the operation of tapping in inflammatory cases, for by resorting to it at an early period the deformity and impairment of lung power, which I have referred to, may be largely diminished.

In such cases, therefore, as I have described, I do not hesitate to tap the chest, and *the existence of pyrexia—even of a high temperature—is no bar to the operation.* The removal of a portion of the fluid is usually followed by a diminution of fever, and other signs of improvement.

I will now pass on to speak of those effusions which are the result of chronic inflammation. Under what circumstances should we tap in these cases? Probably no one would hesitate to perform the operation when one side of the chest was found full of fluid, when the heart was largely displaced, when dyspnœa existed, or symptoms of syncope had occurred. In the presence of urgent symptoms such as these delay is very dangerous. But there may be great danger, when the amount of fluid is considerable, although there does not appear to be any serious interference with the breathing, especially if the patient is somewhat advanced in years or debilitated. Long ago the danger of delay was pointed out by Trousseau, who gives several very illustrative cases. It has been insisted on by others, and I will mention an experience of my own bearing on the subject.

A man was under my care in the Northern Hospital. I had made a diagnosis of extensive

pleuritic effusion on the left side, and had arranged to tap the chest at my next visit should no improvement take place. Two days later I visited the hospital. I learned that, on the morning of that day, the man had died suddenly of syncope. We found a very large effusion in the left pleura.

I think, however, that the operation of tapping is desirable in many cases of chronic effusion when the amount of fluid is not so excessive as in those just referred to. When constitutional measures fail to produce any effect on the disease I think it is better to withdraw a portion of the fluid. It saves the patient's strength, promotes absorption, and accelerates recovery.

Although, however, I advocate tapping in these cases, I admit that very extensive effusions may be removed under the administration of so-called absorbents. I have seen effusions of a very extensive kind absorbed during the exhibition of iodide of potassium and iodide of iron, with the external application of iodine. But the process is slow, and waiting is not unattended with risks. Some cases which apparently present most unfavourable symptoms undoubtedly get well without tapping. Some years ago a young lady was brought to my consultation-room for examination. She had suffered from dyspnœa for a few days. She had been, within a week, dancing at a garden party. I found the left chest full of fluid, and the heart beating under the right nipple ; moreover there was a mitral murmur. The patient had travelled several miles to see me.

I advised her mother, who brought her, to get her home as soon as possible, and I arranged to see her the next day with her usual medical attendant. My own view was to tap the chest without delay, but the patient was very unwilling to submit to the operation, and, as there was a probability that the effusion was recent, we resolved to try the effect of medicines for a time. These produced the desired result. The patient was kept in bed, iodide of potassium and other medicines were administered, and iodine was applied externally. The fluid was gradually, but very slowly, absorbed; had a portion of it been withdrawn by aspiration I believe the recovery would have been more rapid.

I now pass on to consider the question of tapping in passive effusions. Here we must be guided by the same rules as those which influence us in drawing off fluid from the abdominal cavity. If the functions of the lungs and heart are seriously interfered with by the accumulation of fluid in the pleura, we can often give great relief by withdrawing a portion of it by tapping. Sometimes in these cases one side of the chest is much fuller than the other, probably from the fact that some slight inflammatory action has been going on there.

What I have hitherto said applies to effusions which are serous, sero-fibrinous, or sanguineous. I shall refer to the subject of purulent effusion, or empyema, later on. In sanguineous effusion a guarded prognosis should always be given. The presence of blood may be due to the existence of

malignant disease. At the same time I have seen cases where the effusion was decidedly bloody which made a good recovery after tapping.

In performing the operation of tapping we should have clearly in view the effects which may be expected from it. It must be looked upon as an aid to constitutional treatment, and we must not omit other measures for promoting absorption of the fluid. Undoubtedly, in many cases, tapping seems to stimulate the absorbents and favour the action of other remedial agents.

I now wish to say a few words of the operation itself, for the performance of which we possess, in the pneumatic aspirator, an instrument of great value. Although I have drawn off fluid from the chest by means of an ordinary cannula attached to a tube placed under water, without any air getting into the chest, I much prefer the aspirator. It is important that no air should enter the chest during the operation. Although I believe that the entrance of a small quantity of air does no harm, I consider this accident a reflection on the skill of the operator or the condition of his instrument, and the operator is responsible for the latter.

As regards the site for tapping, I prefer, under ordinary circumstances, that recommended by Bowditch. The rule is to find the inferior limit of the sound lung behind, and to tap two inches higher than this on the pleuritic side, in a line perpendicular from the inferior angle of the scapula. There are cases where it is necessary to tap the

patient whilst reclining in bed, and this may necessitate the choice of some other spot.

The size of the cannula used is very important. It should be very small in cases of serous effusion. The use of a small instrument implies a slow withdrawal of the fluid, which is a matter of great moment. In the removal of the fluid the safety of the patient should be mainly considered, and not the rapidity of the operation; and it cannot be doubted that if the fluid is drawn off rapidly, not only may great pain be caused, but also danger is incurred. The lung having been compressed for a longer or shorter time does not readily adapt itself to the altered conditions of the pleura if the fluid is withdrawn rapidly, and the same remark applies to the heart. In my own cases I have always insisted on the use of an almost capillary cannula, and I have never seen any ill results follow the operation. I have known great pain caused by the rapid withdrawal of the fluid, but when it has been drawn off very slowly I have scarcely ever found pain to occur. Much was said some time ago about the copious albuminoid expectoration which is apt to follow paracentesis thoracis. I have never seen really copious expectoration follow any operation in my own practice. I have now performed or directed the performance of the operation a very large number of times, and I have only on one occasion seen expectoration in any quantity follow. In this instance the amount did not exceed half a pint. I cannot help thinking, therefore, that the mode of operating

may have had much to do with this result. Again, the rapid withdrawal of a large quantity of serous fluid is not unattended with danger. Although I have never seen any bad results in my own practice, I have known fatal syncope to follow half-an-hour after an operation in which a large quantity of fluid was somewhat rapidly withdrawn; and this leads me to speak of the quantity which may be safely removed. This must depend much on the amount which the chest contains; but when we have withdrawn fifty, sixty, or eighty ounces, I think it is generally advisable to stop, and repeat the operation in a few days if much fluid remain; in fact, we should not attempt to remove the whole of a large effusion at one operation. If pain or a sensation of constriction across the chest is complained of during the operation, which is often the case, the removal of the fluid should be stopped for a time, and if the pain or sensation become severe and continuous the cannula should be withdrawn. These symptoms are, I believe, often the result of the attempt of the lung to expand, and, as it is covered by a layer of lymph more or less organised, difficulty is experienced and pain produced.

There is one accident which I must refer to which may necessitate the withdrawal of the fine cannula and the substitution of a larger one. Sometimes the fluid is of such a nature that fibrinous coagula form in the cannula, and stop the flow of the fluid which, however, may go on flowing if a larger tube is introduced. I think it is better to use a trochar and

cannula rather than a pointed cannula alone for the operation. There is less risk with the former of wounding the lung; and although this is often covered with a leathery exudation in old effusions, and a slight puncture in such cases would do no harm, in recent cases such coating does not exist, and a wound might be attended with considerable hæmorrhage. Antiseptic precautions may be used in the operation, but I think that carbolising the instrument introduced into the chest is all that is necessary.

Let me now say a few words about the objections which have been advanced against tapping. It is said to convert a serous into a purulent effusion. This is an assertion easy to make, difficult to prove, and equally difficult to disprove. I do not consider it has much weight. Some effusions will become purulent under any circumstances, but I do not think it is ever owing to the operation when carefully performed in the manner which I have advocated. I have repeatedly tapped the same patient several times, and have found the fluid serous to the last. As I have said before, pyrexia is no bar to the operation; the existence of fever at the time it is performed does not necessarily cause the exudation to become purulent.

Phthisis has been supposed to be caused by tapping, but we all know that it is frequently associated with pleuritic effusion, and may develop itself whether the operation is performed or not.

Amongst the other objections raised, are—the

fear of causing syncope, the formation of clots in the pulmonary vessels, and albuminous expectoration. All I can say is that, after a very large experience of tapping, I have never seen any of these accidents when the operation has been performed in the manner which I have described, and with the precautions which I have insisted on, and I consider that I am justified in saying that it may be done without risk.

I will reserve the consideration of the subject of empyema for another lecture.

CHAPTER VIII.

Empyema.

(ABSTRACT OF CLINICAL LECTURE.)

Gentlemen,

The subject to which I wish to call your attention to-day is Empyema, a disease of which we have lately had several instances in my wards.

By Empyema we mean a collection of pus in the pleura. The fluid may be free in the pleural cavity, or it may be in a cavity which is circumscribed by adhesions. Most frequently the former condition obtains.

Empyema arises as the result of acute, or of more or less chronic, inflammation of the pleura. There can be no doubt that at times an acute pleurisy is purulent from the very beginning. I have met with several cases in which a severe attack of pleuro-pneumonia has necessitated the early operation of tapping, and the fluid has been found purulent within a short time of the onset of the disease, thus rendering it highly probably—nay, almost certain—that pus had been poured out from the commencement. Some winters ago I attended, with Mr. Newton, a lady who was attacked with severe pleuro-pneumonia of the right side. Signs

of a large effusion soon showed themselves, and about the tenth day of the illness we tapped the chest and removed a large quantity of thick creamy pus. Again, I have quite recently attended a lady in whom thick creamy pus was found within three weeks of an attack of acute pleuro-pneumonia.

Cases of this kind, where the pleurisy is purulent from its onset, are more frequently met with in children than in adults, but they are by no means infrequent in the latter.

An acute effusion, which is serous at first, may become purulent, after no great lapse of time. I have tapped cases where I have drawn off serum and the fluid has re-collected, and at a subsequent tapping has been found purulent. Quite recently I attended a gentleman who had a violent attack of pleuro-pneumonia, with signs of consolidation of the lung and effusion. On the twelfth day of the disease the chest was punctured, and serum was drawn off. The symptoms did not improve, the temperature remained high, and at the end of a fortnight the chest was again tapped and pus was discovered. A drainage-tube was inserted, and the patient made a good recovery.

In cases such as this, where, on a second, third, or subsequent tapping, the fluid, previously serous, is found to have become purulent, we must not attribute the change which has taken place to the operation, unless the latter has been badly performed and air has been allowed to enter the chest freely. I have frequently tapped the same patient

several times and found no alteration in the character of the fluid, which has remained serous throughout.

The most numerous cases of empyema, however, are those which are the result of chronic inflammation of the pleura, the effusion either being purulent from the first, or subsequently becoming so, having been originally serous. The latter mode of origin can undoubtedly occur, for occasionally we meet with cases of chronic effusion where, at the first tapping, we find serum, but after a time, when we have occasion to repeat the operation, we find pus. Moreover, whenever there is a strumous diathesis, there is a tendency for inflammatory processes to become purulent, and we know well that phthisis is often associated with empyema. I also think that the prolonged presence of a serous effusion in contact with the pleura may cause such changes in that membrane as to lead to an effusion of pus, a result which would not have taken place had the fluid been removed earlier.

I will now say a few words on the subject of diagnosis.

The physical signs of empyema are those of fluid in the pleura. There is nothing special in them, but the general symptoms differ somewhat from those of serous effusion. The temperature is often higher; there may have been rigors, but these do not always occur. The patient's general condition is not good. There may be night sweats and emaciation, with high temperature in the evening,

and in some cases œdema of the chest walls is seen. You must not, however, rely too much on the general symptoms, but if, from a consideration of these and the physical signs, you suspect the presence of pus in the chest, you must make an exploratory puncture in the manner I have described in a previous lecture, taking care to use a cannula sufficiently large to ensure that pus will pass through it, for I have often seen that when a fine cannula has been introduced, nothing has been withdrawn, whereas, on repeating the puncture with a larger cannula, pus has appeared; but in some instances I have found that nothing could be withdrawn without the use of the pneumatic aspirator. As, however, I have dwelt at some length elsewhere on exploring and tapping the chest, I need not further refer to the subject now.

Having satisfied yourself that the fluid in the chest is purulent, what is the best treatment to adopt?

When once an empyema has formed, it is probable that, as far as regards adults, the pus is never absorbed, and it is useless to attempt to promote its absorption by constitutional measures such as might be beneficial in a serous effusion. In children, however, it is quite possible that absorption may take place. Further, we know that in them recovery may follow after one or more tappings, without recourse to further proceedings. Possibly in such cases the collection of pus is circumscribed. In children, therefore, when you have established your

diagnosis you may tap, and then wait to see the result. Possibly no further accumulation of pus may take place, but should it re-accumulate you must tap again, and then you must consider whether or not the process which I shall refer to as regards adults should be resorted to.

With reference to the treatment in adults, I may say that I have never seen a case where tapping alone has been followed by recovery. The oldest case in which I have seen it succeed was that of a boy aged seven years, about whom I was consulted some years ago. He was tapped, and about half a pint of pus was drawn off. After an interval of four weeks he was tapped again, and about five ounces of pus were removed. No further accumulation took place and he perfectly recovered.

The only effectual way of dealing with empyema in adults consists in establishing a free external discharge, and this is best effected by the insertion of a drainage tube. The plan I prefer is, to pass a good-sized tube through an intercostal space, and bring it out through a counter-opening in a lower space. The tube should be introduced as low as possible in the chest towards the posterior part, due allowance being made for the arch of the diaphragm, and for the contraction of adhesions between it and the chest walls during the falling in of the latter, which will take place in the process of recovery. In recent chronic cases, I think it is desirable to draw off the pus, and then wait for its re-accumulation before inserting the tube. This

gives the lung a chance of expanding; but in older cases it is so covered with organized lymph that often but little, if any, expansion can take place. In acute empyema it is desirable to operate as soon as the diagnosis is established. When we have to deal with acute pleuro-pneumonia, causing empyema, there is generally some serious impairment of the general health, and such cases are apt to do badly, but an early resort to operation, and the insertion of a drainage tube, may be the means of saving them.

I prefer the operation I have described to that of inserting a tube by a single opening and draining off the fluid by siphon action. This latter process possesses the advantage of not admitting the air freely into the pleural cavity, and it may be successful in some cases; but it necessitates the patient's remaining in bed, and I have seen it frequently fail. In one instance the plan was carried out for me very carefully by Mr. Paul. The patient was tapped with a large trochar and cannula, and through the latter a drainage tube was passed into the chest, the other end of the tube being placed in a vessel containing carbolic lotion standing by the side of the bed. The fluid drained into the vessel. After a time—some weeks—it became serous, and soon it nearly ceased to flow. There was good resonance over the chest, and it was quite evident that the lung had expanded, and I hoped it had contracted adhesions with the chest walls, and that a satisfactory recovery had been

made. The tube was consequently withdrawn, and the opening closed. After the lapse of a short time, the temperature began to rise, and the effusion returned. The chest was again opened, pus was found, and a drainage tube was inserted in the usual way. The patient recovered. I have known a similar result follow in other cases when a single opening has been made and the siphon plan of drainage carried out, and I cannot therefore recommend it for general adoption, but I think there may be cases in which it is worth trying.

There is always danger incurred in delaying the tapping of an empyema. The lung becomes so coated with lymph, and so much constricted, that the chances of its ever expanding diminish from day to day. And it is this want of expansion of the lung in old cases that causes the operation in empyema to be so frequently only partially successful; the truth being that the lung is collapsed and compressed at the back of the chest, and but little or no expansion takes place, the patient practically living on with only one lung. There may be also great falling in of the chest walls, and considerable curvature of the spine. There is, moreover, another source of danger in delay. The lung may rupture, and a communication be established between the pleural cavity and the bronchial tubes. This is shewn by the patient expectorating a large quantity of pus, and in some cases recovery has followed this mode of discharge of the fluid. But independently of the fact that the passage of the pus

through the lung may so disorganise it as to lead to a fatal issue, the expectoration of the matter is very distressing to the patient, and the process is often very exhausting. Whenever, therefore, this accident occurs, you should at once insert a drainage tube through the chest wall, in the way I have described, and the free exit of the fluid through this channel may cause the opening in the lung to close.

I need not insist on the importance, both as regards the operation and the after-treatment, of antiseptic precautions and extreme cleanliness. In many cases, especially in hospital practice, and unless the greatest care is taken, the discharges become fœtid. This may necessitate the washing out of the pleural cavity. In doing this, let me warn you not to use carbolised fluid. I have seen very severe symptoms produced by it. I think it is safer to use Condy's fluid or pure water. In the after-management of a patient who has been submitted to the operation, a careful record of the temperature should be made. Any rise in the latter almost certainly indicates a retention of matter, and this may exist although apparently the discharge is free.

You must be guided by circumstances as to the length of time you leave the tube in the chest. If there is no expansion of the lung it may be left in indefinitely. I have had patients who have gone about for a long time with a tube in the chest, and one lived with it in for several years. In other

cases where the discharge gradually diminishes, and ultimately almost entirely ceases, and where you have evidence from physical signs that the lung has expanded and probably contracted adhesions with the chest-walls, you may remove the original tube and introduce a smaller one, and this after a time may be replaced by one still smaller, or by a thread, until, feeling satisfied that nothing but a mere sinus remains, the thread also may be withdrawn.

I have never had a case where it was necessary to remove a portion of a rib in the operation, but in old standing cases, where the ribs have come into close contact with each other from extreme contraction of the chest-walls, such a process may be desirable. When once a tube has been inserted through an intercostal space, I have never known it become closed by the approximation of the ribs between which it was placed. I believe that the pressure of the tube causes absorption of the bones where they are in contact with it, and thus the opening is preserved. In a case referred to by Mr. Banks this condition was found after death.

In a now somewhat extensive experience of cases of empyema I have met with two cases, and two only, in which abscess of the brain formed and caused death. Both cases were in No. 10 ward. Both had been tapped, and a drainage-tube had been inserted in the usual way. After a time convulsions of an epileptiform character occurred, and death took place after several weeks. A single abscess was found in each case in the brain. It

was doubtless pyæmic. The possibility of such a result as this should impress on you the importance of great care in the management of your patients after operation.

In conclusion, I would remark that, although in some cases of empyema we get satisfactory results from the mode of treatment which I have mentioned, in others, either from the collapse and constriction of the lung due to the causes I have referred to, or from collapse due to free admission of air into the pleural cavity when the chest is opened and subsequently, the results are disappointing; the lung not expanding at all, or only to a slight extent. I think our present mode of procedure might be improved. We want a method of operating by which a tube of sufficient size could be introduced into the chest and brought out through a counter opening, so as to establish free drainage, without admitting air so freely into the pleural cavity as to cause collapse of the lung. Under these conditions the lung would have a better chance of expanding as the fluid drained away, and before long would be safe from the danger of collapsing by the formation of adhesions to the chest walls. Some of the most satisfactory recoveries that I have seen have been in cases where the empyema has been the result of pleuro-pneumonia with consolidation. In such cases the lung cannot collapse when the chest is opened, and adhesions are soon formed between it and the chest walls and thus the pleural cavity becomes obliterated.

CHAPTER IX.

On Cases of Perihepatic Abscess bursting through the Right Lung.

(CLINICAL LECTURE.)

Gentlemen,

I wish this morning to call your attention to the case of Charles O., who was in No. 10 ward, and who suffered from a very unusual form of disease. There is another case of a similar kind, which occurred in my practice at the Northern Hospital, to which I will also allude.

Case I.—The man, C. O., was the subject of a perihepatic abscess; an abscess forming not in the substance of the liver, but behind it; and which had found its way through the diaphragm into the right lung; the matter from it becoming expectorated. I need not tell you that cases of this kind are very rare. Abscess of the substance of the liver is more common. In this country, however, it is scarcely ever seen, except in those who have been in tropical climates and have contracted the disease there. What are called pyæmic abscesses are more frequently met with. A man has pyæmia, and abscesses may form in any part of the body—in the lungs, in the liver, or elsewhere. But the abscess which results from hepatitis or perihepatitis is generally of tropical origin. The man of whose case I wish to speak first was twenty-nine years of age. He had followed the occupation of carter, but he had previously

been in the army, and had served in tropical climates. He had suffered twice from dysentery—and I wish you to bear this fact in mind—once in 1858, and again in 1870; on the latter occasion the disease lasted about a month. He was admitted into the Infirmary, under my care, on December 18th, 1875. He told us that his illness began six months before admission with pain in the right side, and cough. He was confined to his bed for five weeks, and then he remained fairly well for six weeks, when, his symptoms returning, he was admitted into the Infirmary, under one of my colleagues. He remained here till Nov. 6th, and during his stay he spat up some blood. He was re-admitted under my care, as I have already said, on December 18th. The day before admission he spat up, he said, a large quantity of blood. The family history of the man was good, and I could find no indication of constitutional taint, tuberculous or syphilitic. He was much emaciated, very weak, pale, and somewhat sallow, but there were no night perspirations, and no jaundice. The temperature was 100 deg., the pulse 120. On examination, we found the tongue red and somewhat furred; the bowels were loose. He complained of severe pain on the right side over the liver, increased on inspiration, and a constant pain in the right shoulder, and there was also pain on percussion in the infra-axillary and lower dorsal regions. The area of hepatic dulness was slightly increased upwards behind. The urine was normal. There was dulness, not very marked, over the back of the right lung, beginning at the lower angle of the scapula. Towards the extreme base, the dulness was more decided. Puerile breathing was heard over the whole of the left lung and the front of the right; with bronchial breathing, coarse crepitation, and bronchophony over the lower part of the latter below the inferior angle of the scapula. Over a large portion of the right lung bronchial vibration was felt, indicating the

presence of a good deal of fluid in the bronchial tubes. The expectoration was of a peculiar character; it was copious, somewhat frothy, bloody, purulent, and rather tenacious. It had somewhat the appearance of the sputum met with in some cases of pneumonia with gangrene, but it was not fœtid. There was a peculiarity about the mode of expectoration; the sputa were not ejected as they usually are in pneumonia, but in large mouthfuls, and at times in large quantities, with long intervals between the ejections.

Now, what was the disease from which this man was suffering? Was it pneumonia of a low form terminating in abscess; or phthisis, with a cavity into which blood and pus were being poured? Or was it an abscess connected with the liver, which had burst into the lung? There was a history of dysentery, and the man had been in a tropical climate, circumstances which might induce some disease of the liver. Having already seen two cases, to which I will shortly refer (one of which terminated fatally), where the symptoms had been very similar to those which I have detailed, I did not hesitate to express an opinion that the man was suffering either from an abscess of the liver, or one that had formed between the liver and the diaphragm, and had burst into the lung. I inclined most to the latter view.

As regards the treatment of the case, I may say that the man was put on quinine and acids with a moderate amount of stimulants.

I have alluded to the way in which the patient expectorated, and I find a note to the effect that the

sputa were brought up at intervals, and in large quantities. The man would cease to expectorate for many hours—twelve, and in one instance twenty-four—and then, all at once, he would cough up a large quantity of bloody purulent matter. Evidently there was a cavity, the communication between which and the main bronchial tubes became occasionally closed; the cavity then filled, and copious expectoration followed. The temperature of the patient never rose very high. On December 19th, the pulse was 120, and the temperature 100·7 deg., and, I believe, it never rose above 101 deg.

I need not dwell long on the progress of the case. The man became gradually worse; he continued to expectorate the same kind of matter almost up to the time of his death, except that on some days it was free from discolouration. The sputa were examined to see if any lung-tissue were present; but I said that, the abscess having burst into the lung, the latter would be disorganised, and yellow elastic tissue might be discovered in the expectoration. This was found to be so, but it did not at all alter my opinion of the case. We gave the man opium to relieve the pain, and continued the quinine, and acids, and stimulants, with as much food as he could take. My object was to carry the man on so that the abscess might discharge itself and close; and I hoped that by supporting him well, this result might possibly be accomplished; but the disorganisation of the lung, as revealed at the post-mortem examination, was too great to admit of

recovery. The patient died on January 7th, 1876, and the necropsy was made on the same day; and before I read the notes of it, let me again remind you that the man had suffered from dysentery on two occasions.

Necropsy.—The left lung was very large and emphysematous, filled with frothy mucus, and non-adherent. The right lung was adherent by its lower lobe; between it and the liver there was a collection of muco-purulent sanious matter; the cavity containing this indented the right lobe of the liver, forming a depression three inches in circumference and one inch deep. It was lined with a membrane nearly one-eighth of an inch thick. The substance of the liver was not involved. The abscess thus situated, was continuous, through the diaphragm, with a large cavity in the lower lobe of the lung filled with muco-pus, and communicating with the bronchial tubes. Portions of lung at the surface were œdematous. The kidneys and spleen were healthy. The colon was contracted; its muscular coat was thickened, and its mucous membrane puckered, congested, and thickened. The mesenteric glands were enlarged. The small intestines were slightly dilated.

In this case, an abscess had formed at the back of the liver, not involving its substance, and had found its way through the diaphragm into the right lung.

Now let me refer you to another case of a similar character.

Case II.—Louis St. L., a ship's cook, was admitted into the Northern Hospital, under my care, on October 27th, 1870. He had come from the Coast of Africa, and had suffered from pain in the right side for three or four months. He had been spitting blood he said for about a month, but

previously there had been no cough. No doubt at the time the cough and hæmoptysis occurred, the abscess which the necropsy revealed burst into the lung. The man was much emaciated and very weak when admitted; and he was bringing up a large quantity of sputum, similar to that which I have described in connection with the last case; it was reddish, muco-purulent, and somewhat frothy, but free from fœtor; it was expectorated in large mouthfuls, and easily. On examination, we found a fulness at the lower part of the right side of the chest, and there was some crepitation over the middle of the right lung behind; but at its base the respiratory sounds were inaudible. The pulse, on admission, was quick; the temperature was not taken. There was no jaundice.

I must confess I was in doubt as to the nature of the case. Was it a low form of pneumonia, which had terminated in abscess? Or was there an abscess commencing elsewhere, which had found its way into the lung? Had the case commenced as hæmoptysis which had been succeeded by pneumonia? Brandy and milk, with gallic acid and tincture of opium were ordered.

On October 28th, the pulse was 110, and the temperature 98·5 deg.; and on the following day the pulse was 120. There was great pain in the right side; the physical signs were unchanged; the sputum was very copious. There was no material change in the patient's condition till November 1st, when he began to pass blood by the bowels. He died on November 3rd.

Necropsy.—The body was much emaciated; the right lung was adherent in front and strongly so behind; it was pneumonic at the extreme base. The bronchial tubes were filled with sanguineo-purulent matter. There was an aperture at the lower part of the lung communicating with an abscess. The left lung was adherent. *Liver.*—The greater part of

the tissue appeared tolerably healthy; behind, the capsule was greatly thickened; between it and the abdominal wall, beside the ribs, there was an abscess communicating with the right lung through an opening in the diaphragm. *Intestines.*—The colon was greatly thickened, especially towards the sigmoid flexure; numerous ulcers were present. No communication existed between the abscess and the bowels.

These two cases very closely resemble each other in their history, their course, and their post-mortem appearances. I have seen two other cases where the symptoms were very similar to those which I have described, and in which, I believe, an abscess connected with the posterior part of the liver had burst into the lung. One case terminated fatally, but no necropsy was obtained; in the other the patient recovered; so that in neither instance was it possible to verify the diagnosis which was made.

Case III.—I was summoned in 1873 to see, in consultation, a gentleman who had had some symptoms supposed to be due to pleurisy. A friction-sound was heard in the lower part of the right chest. He did not, however, improve under the treatment recommended; and at the time I saw him, he had begun to expectorate a good deal. He had been in India, and had been told that he had disease of the liver. He had suffered from pain in the right side for many months, nay, even some years, he said. On examination, I found some dulness with crepitation at the base of the right lung, but no signs of lung-disease elsewhere. There was a good deal of sputum, which resembled that of the two patients whose cases I have detailed. Looking at the history of the patient and the peculiar character of the expectoration,

my diagnosis was that he was suffering from hepatic or perihepatic abscess which had opened into the lung. I saw the gentleman two or three times. The expectoration continued of the same character, and the general symptoms remained for some time severe. He ultimately recovered.

Case IV.—Again, I saw in consultation, in the spring of 1875, a lady, living in the north of Lancashire, who had symptoms very similar to those of the last case. She had been ill for four or five months. She had had symptoms of disordered liver. There had been a slight attack of a dysenteric nature. She had never been in a tropical climate. She had suffered from cough; and had, on more than one occasion, brought up a large quantity of sputa. A pleuritic rub had been heard in the right pleura; and there had been dulness over the lower part of the right back. The case had been looked upon as one of lung disease. When I saw the patient, she was pale, sallow, but not emaciated. There was a severe hacking cough, with expectoration which was reddish and free from fœtor. It was very similar to that of the other cases I have referred to. The pulse was 92. There was no evidence of disease at the apex of the lung; nor were there any general symptoms of phthisis. The point for consideration was whether the case was one of abscess of the lung, or of abscess connected with the liver which had burst into the lung. My opinion was in favour of the latter view. The patient died about a fortnight after I saw her, but no post-mortem examination was made.

These four cases presented, during life, symptoms and physical signs of a very similar character. Three of them, when seen in their earlier stages, were considered to be cases of chest disease; nor would it have been possible, perhaps, before the abscesses had burst, to say that the pleuritic rub or

the dulness at the base of the lung, was not due to primary disease within the chest.

In cases of this kind, what is the proper treatment to adopt? It is most important that a correct diagnosis should be made, so that you should not be treating as a primary disease of the lungs one which is only secondary. Your object must be to support your patient well, in the hope that the abscess may ultimately close. The bursting of an abscess connected with the liver into the lung, and the discharge of the matter through the bronchial tubes, is not the most satisfactory mode by which such an abscess may make its exit, because the lung may become so disorganised during the process that death may result. If an abscess burst into the colon, for example, and the discharge take place through the bowels, the latter may not become at all disorganised, and the chances of the patient's recovery are greater than if the abscess made its way into the chest.

Could anything be done in such cases as I have referred to, as regards opening the abscess or drawing off its contents? It is quite clear that, when an abscess forms in the situation of those which we discovered on post-mortem examination, it could not be reached. Indeed, you could not make an accurate diagnosis of its seat, even if you surmised its existence before it had burst. All, therefore, that you can do is to rely on constitutional measures.

[In connection with the subject of this lecture, I

will refer to a very interesting case of biliary fistula connected with the lung which I saw some years ago. The patient was a lady under the care of Dr. Cameron, with whom I saw her. She was, and had been for a considerable time, expectorating a large quantity of bile. There was no definite history as to the exact cause of the fistula, but the probability was that there had been a hydatid tumour of the liver, which had opened into the lung. The lady lived for some years after the formation of the fistula. At times the discharge of bile ceased, but only to recur.]

CHAPTER X.

Alpine Winter Climates.*

A recent visit to Maloja in the Engadine and to Davos-Platz has enabled me to make an investigation of these places, and to form an opinion, from personal observation, of their value as health resorts, and their suitability as winter residences for patients suffering from pulmonary consumption and other affections; and I have thought that a few remarks on the subject might not be without interest to the members of this Society.

My visit was paid during the past summer (1886), in the months of July and August—in what may be called the height of the summer season; and I remained both at Maloja and at Davos sufficiently long to enable me to make a tolerably complete investigation of both places, and to note the important features of each as regards the special points to which I shall direct attention.

First, as to Maloja :—

The Engadine, as many of you are aware, is a valley about sixty miles in length, running N.E. and S.W., and enclosed by mountains of consider-

* "Notes of a visit to Maloja in the Engadine and Davos-Platz, with Remarks on the Suitability of these Places as Winter Resorts for Patients suffering from Pulmonary Consumption and other Diseases."—A Paper read before the Members of the Liverpool Medical Institution, and re-printed from the *Liverpool Medico-Chirurgical Journal.*

able height. The altitude of the valley varies from about 3400 feet to 6000 feet. What is called the Upper Engadine is that part which lies between the Maloja—its extreme upper end—and the village of Samaden. The former is about 6000 feet, the latter about 5700 feet, above the level of the sea. The distance between the two places is about fifteen miles. In this district are situated the New Maloja Hotel, the villages of Sils and Sils Maria, Silvaplana, Campfer, St. Moritz, Celerina, and Samaden; whilst nearly opposite Samaden is Pontresina, situated at the entrance to a valley running off at a right angle to the larger valley of the Engadine. Along the centre of the valley runs a chain of lakes, and the river Inn.

As is well known, this district has become of late years not only a very favourite health resort during the summer season, but also a winter resort for patients suffering from consumption, the idea being that the purity of the atmosphere, the absence of microbes, the bracing effects of bright sun, and the general invigorating influence of the mountain climate might beneficially affect the disease, although the temperature is exceedingly low, and snow remains on the ground for many months.

But another place which has had a still greater reputation for consumptive patients as a winter resort is Davos-Platz, situated not far from the Engadine, having a climate somewhat similar to it, but with certain differences which I shall refer to by and by.

First of all I will speak of Maloja, a place which has quite recently been brought into notice in consequence of the erection there of a magnificent hotel, and of the publication of a work by Dr. Tucker Wise, the resident physician of the establishment—a work which has been circulated very extensively amongst the profession throughout Europe.

The Maloja Hotel is situated at the extreme end of the Upper Engadine, within about ten minutes' walk of the summit of the Maloja Pass. From the summit the road descends in a series of zigzags very rapidly towards Italy; so that in a short drive a descent of more than 2000 feet—to Promontogno—can be made, and if the journey be a little more prolonged, Chiavenna, 5000 feet below Maloja, can be reached—a not unimportant point with reference to certain invalids for whom a quick change to a warmer spot might be desired. The Maloja Hotel, recently built, was opened two years last August (in 1884). It can accommodate about 350 visitors. It is a very fine building, very handsomely fitted up, furnished in a very elaborate style, with large dining-rooms, reception-rooms, etc., and a very handsome concert-room, where excellent music is provided daily for the inmates. The bed-rooms are good, and comfortably furnished. The cuisine is excellent, and, in fine, the hotel will compare most favourably with the best class of Swiss hotels, which are amongst the best in Europe. As a summer residence it affords great attractions; but it is attempted to make the hotel a winter resort for

invalids, and it has been built with that object in view. In order to keep out the cold, the windows are all double; and to warm the interior of the hotel, a special apparatus of a very elaborate kind has been constructed, at a cost of about £20,000. On the basement floor are several so-called batteries, which are heated, and air drawn from outside, therefore pure, is warmed in them, and then circulated in air-shafts throughout the house.

In each bedroom close to the floor in one corner is a small sliding door, communicating with the hot air-shaft; by opening this door when the batteries are at work hot air is admitted into the room, and there is a ventilating shaft in another corner of the room, which has near the ceiling a valve connected with it, by opening which the air is carried out of the room. A similar plan is adopted in the sitting-rooms. That is, in brief, the system by which the hotel is heated and ventilated. There are no stoves, and no open fire-places. There is an apparatus by which ozone is generated, and can be distributed to certain parts of the hotel.

During my stay at Maloja we had an experience of wintry weather. We had on the 27th of July a severe snowstorm, and another of less severity a few days later. The weather was very cold. The batteries had to be put to work, and I can testify to the fact that the rooms became very warm. The drawing-room reached 70 deg. Fahr. To my mind the heat was by no means as pleasant as that diffused by a fire. In the winter months the

batteries are set at work about seven o'clock in the morning, and before patients wish to rise the air of the hotel becomes warmed, and the bedrooms can be warmed to almost any temperature the occupants please by regulating the action of the sliding door which admits the warm air.

The outdoor amusements during the winter months are skating—for which a rink has been made—tobogganing, sleighing, walking, etc.

I may remark that for the purpose of heating the batteries, and for cooking, coals are used, and inasmuch as these are brought from Belgium the cost is very great, each ton of coals delivered at the hotel costing from 75 to 80 shillings; and I was told that during the winter season the cost of fuel for the house was about £8 to £10 per day. Last year (1885-6) about forty patients passed the winter at Maloja, this year more are expected.

I will leave what I have to say about the climate of Maloja and the Upper Engadine till I have spoken of Davos-Platz.

The valley in which Davos-Platz is situated is about ten or eleven miles long and half a mile broad. It is enclosed by moderately high mountains, and there are pine forests close to the village. Its elevation is about 5000 feet above the sea, about 1000 feet lower than Maloja. The actual height of Davos, according to Baedeker, is 5115 feet. The valley, like that of the Engadine, runs N.E. and S.W., and is separated from the Lower Engadine by a mountain range. Davos itself consists of a village of

considerable size, with numerous hotels, and one is surprised, on first coming in sight of it, to see how much like an ordinary watering-place it looks. There are ten resident medical men. The popularity of the place has increased very much during the last few years.

The subjoined table shows the greatest number of patients with their friends, who have been at any one time in Davos, in each of the twenty years since Davos became a health resort. (The total number of those who visited Davos during any one of these years is, of course, much larger, and as regards them the figures in the table cannot even be called approximative.)

Years.	Patients.	Years.	Patients.	Years.	Patients.
1865	2	1872	200	1879	770
1866	12	1873	300	1880	855
1867	25	1874	400	1881	920
1868	50	1875	500	1882	1015
1869	70	1876	550	1883	1050
1870	90	1877	560	1884	1300
1871	120	1878	700	1885	1300

Whilst at Davos I visited almost all the hotels, in company with one of the resident medical men. They are all very comfortable, and some very handsomely fitted up. The largest is what is called the Kurhaus, much frequented by Germans. Other hotels, such as the Belvedere, Victoria, Buol, are chiefly patronised by the English. At the Belvedere, where I stayed, the landlord endeavours, and very successfully, to meet the wants and wishes of his English clients.

The village or town of Davos is situated on the north-west side of the valley in which it lies, and therefore has a south-east aspect. Through the valley runs the Landwasser River—a rapid stream. Near the principal hotels a large skating rink has been made.

A few words are necessary about the drainage of the place, as well as of that of Maloja. At Maloja the hotel is drained into the lake of Sils, which is only a short distance from the hotel. The sewage is carried well out into the lake, and the general sanitary arrangements of the hotel are very good. At Davos, a few years ago, a great outcry was made about the drainage, and some expensive sewerage works were set on foot. These are now completed. The hotels are well supplied with water-closets, and the sewage is carried into the Landwasser River far below the village,

As regards the heating and ventilation of the hotels at Davos. One of these, the Kurhaus, the hotel largely frequented by Germans, is heated by hot air. The other hotels are heated by stoves and open fireplaces. There is in each bedroom a stove, and in the sitting-rooms stoves and open fireplaces. At Maloja coals are burned, and great volumes of smoke are sometimes seen issuing from the main chimney. At Davos the fuel is wood. This is burnt in order that the atmosphere may be as little as possible contaminated by particles of unconsumed carbon. And in a place where there is so little wind, this is considered important both to

the comfort and well-being of the patients. At Maloja the lighting is by electricity, except in the bedrooms, where candles are used. At Davos some of the hotels are furnished with the electric light—in one this is carried into the bedrooms. Further, the people of Davos have this year carried out works, so that the town itself will be lighted in a similar way. The course of the river running along the valley has been diverted through large pipes, and the water-force thus obtained is to drive a dynamo, which will supply the electricity. It is expected that this means of lighting the place will be in operation this winter (1886–7). Another point to be mentioned is that it is now decided to extend the railroad from Landquart to Davos, and it is said that the road will be ready for traffic in 1889. The effect of this will probably be injurious to the place in some respects, but beneficial to it in others.

I will now refer to the general features of the climates of Maloja and Davos. The climate of the Engadine is described as resembling that of Lapland, and the inhabitants say that they have during the year nine months winter and three months cold weather. Certain it is, that whoever goes to the Engadine, even during the summer, should be prepared for a low temperature. One great feature of the climate of Maloja, and indeed of the whole of the Engadine, is the prevalence of wind. The truth is that the air is drawn along the valley, and it is rare in the summer to find the air calm. In my opinion, this circumstance makes the

climate very trying for many people, especially for invalids. To the healthy and robust, and to patients suffering from certain ailments, this does not apply; but it is especially applicable to elderly people, or those whose circulation is feeble.

I must here, however, remark that it is said that during the winter months, especially after the snow has fallen, and except during storms, there is but little wind, and there is bright sunshine, and the meteorological observations of two years tend to bear out this statement. At the same time, I think that, for calmness of atmosphere, the place cannot be at all compared with Davos, where, on a very large number of days in the winter, there is almost complete absence of air movement. The Maloja is remarkably free from mist, and the air is very dry. You may see the mist rising from the valley on the Italian side, and appearing at the head of the pass, and looking like the steam issuing from a huge cauldron, but it is dispersed before it reaches the hotel.

And now as to Davos. One of the most striking features of the valley is the great calmness of the air. In the afternoon perhaps a gentle breeze springs up, but this soon subsides; and the absence of wind, which is even more marked in the winter than in the summer, enables patients to be out in the sun without any sensation of chill. This, together with the purity of the air, which is, of course, shared by that of the Engadine, constitutes the great attraction, and is the most important

curative element of the place. Patients who have resided here during the winter say that they are able to sleep with their bedroom windows partially open.

I doubt if Davos is as free from mist as Maloja. During my stay at the former place one day was decidedly misty.

I will now refer to the detailed meteorological observations of Davos. For some years past the most careful observations have been made, and I have the reports from 1879 to March of the present year. Taking the weather from October to the end of March, and especially in December, January, and February, I find that the great characteristics of it are—(1), a clear, bright, almost cloudless sky, with a warm sun; (2), a very low shade temperature; (3), an absence of wind; and (4), from time to time heavy falls of snow.

Detailed observations of a very careful kind have been made at Maloja for a few years past by Dr. Tucker Wise. The general features of the place are similar to those of Davos. There is, however, as appears from these reports, more wind there than at Davos.

There is another place, Wiesen, not very far from Davos, situated in the same valley, about ten miles lower down, and more sheltered than Davos, with a very calm atmosphere. I should say this place was more suited for cases of phthisis which are somewhat advanced, than either Davos or Maloja, but there is only one small hotel, and the accommodation therefore is very limited.

I now pass on to say a few words about the suitability, as winter residences, of these places for cases of consumption and other diseases, and first of all I will mention the affections and conditions which I think are not likely to be benefited by such residence.

1. Cases of advanced phthisis.
2. Cases of emphysema of the lungs.
3. Cases where there are old pleuritic adhesions.
4. Cases of valvular disease of the heart, and of fatty disease of the heart if at all advanced.
5. Cases of disease of the great blood-vessels.
6. Cases of renal disease.
7. Cases with strong rheumatic tendency.
8. Patients advanced in years, unless their circulatory system is very sound, and even then, I think, these climates are not the best for them.

Now as to the cases which are likely to be benefited. First let us consider pulmonary consumption. That many cases of this disease have benefited largely by wintering in Davos and other Alpine resorts there can be no doubt, and twenty years ago I recommended such places in appropriate cases, but I must express my very strong conviction that cases are sent there in which, instead of good, harm has resulted, and the process of lung disorganisation has been hastened instead of retarded. It is claimed for Davos that cases in which hæmoptysis has frequently occurred, which have in fact a hæmorrhagic character, often do well there; but I think great care should be exercised in the selection of such

cases before sending them. The cases of phthisis, which, in my opinion, are most likely to derive benefit from wintering in Davos, are those where the disease is in a very early stage, where the temperature ranges are slight, where the changes in the lung tissue are slow, and where the general powers of the system are good. I think that, in such cases, not only great improvement may take place, but a perfect recovery may result, by prolonged residence in the locality.

It is not easy to define exactly all the cases which one would consider suitable or unsuitable for Davos, for there are peculiarities in every case which may determine the decision one way or the other; but I should not, as a rule, advise patients to be sent there whose disease is at all advanced. At the same time, it is said that even such cases derive, if not real benefit, great ease and comfort from breathing the pure air of the place.

But I think there are other cases besides those of certain forms of phthisis for which a residence in the winter at Maloja or Davos is likely to be of great advantage—cases where there is no organic disease, but where, from over-work or other causes, great nervous exhaustion and general want of tone have supervened. In such cases, a month or two, or a whole winter, at Davos may be most beneficial. After a time exercise might be taken, and the vigour of the whole system might be restored by the healthy and exciting pastimes of skating, tobogganing, &c., which might be largely indulged in.

Nearly the whole of the day, during sunlight, of probably three days out of four on an average, might be spent in the open air. Indeed, it has now become a practice with some people to take a winter holiday at Davos or St. Moritz for the purpose of enjoying the healthy and stimulating pastime of skating. To such as desire this, I can recommend either Maloja or Davos.

A word as to the time when patients should be sent. My own opinion, confirmed by that of the Davos physicians, is that they should go early in the autumn, in September, or certainly not later than October. By going thus early they have time to get acclimatised before the severe cold sets in. Dr. Wise, of Maloja, however, says he prefers patients not to go there before December, as he says November is often a very bad month, and he thinks it is best for them to keep at a lower level till it is over. Then, as to the time when patients should leave. During the melting of the snow these places are very uncomfortable, and therefore many patients leave early in April. If they remain they must of course stay much indoors for a time. Some patients remain the whole year at Davos, except for July, August, and part of September, when they go to the Engadine or elsewhere just for a change. One of my patients, who is suffering from chronic phthisis, and who went to the Riviera three years ago, wintered in Davos last year, and is wintering there this year. I saw him in August last at Maloja, and subsequently at Pontresina. He spoke very

highly of Davos, and said he had derived more benefit from it than from the Riviera. He had in the early part of his illness a severe attack of hæmoptysis.* Another patient, whom I sent to Davos about nine or ten years ago, who had the early general symptoms of phthisis, but no decided physical signs, derived the greatest benefit from wintering there, and has remained perfectly well ever since.

I think there is one point of importance with regard to residence in the hotels at Davos. They are in winter very full, and a large proportion of the patients are phthisical. They all congregate together in the sitting-rooms, and it cannot but happen that the air of the rooms becomes somewhat vitiated, whatever care may be taken. Now, if there is any truth in the bacillar theory of phthisis—if it is possible to take the disease by inhaling air containing tubercular bacilli—there is some risk to the healthy, and to those suffering from some non-tubercular disease, of becoming infected under the conditions I have named. I have no very strong belief in the theory of the infectious nature of consumption; but I have known of a few cases in my experience where there was strong presumptive evidence that the disease had been communicated from husband to wife, or from wife to husband. It is most difficult to arrive at any definite conclusion on this point; but I have met with a few instances where it seemed highly probable that there had been

* This patient is since dead. He died at Davos.

direct infection from individual to individual. I should advise that all patients, whether consumptive or otherwise, should have sitting-rooms of their own. Of course this materially increases the expense, but it must also add much to the comfort.

It is claimed for the Maloja Hotel that the system of ventilation is so good that the air in it always remains pure, but of course much depends on the care with which the ventilating apparatus is regulated. At present the number of people in the hotel during winter is not sufficiently great to affect injuriously the atmosphere of the public rooms.

TABLE OF CASES

OF

ACUTE PNEUMONIA

WITH ABSTRACT OF TREATMENT

REFERRED TO IN CHAPTER II.

N.B.– Where the quantity of a stimulant is given, it refers to that administered during twenty-four hours, unless otherwise stated.

When Carbonate of Ammonia is referred to as having been administered, it means that it was usually given in 5-grain doses every three or four hours.

Table of Cases of Acute Pneumonia, with Abstract of Treatment.

No.	Name.	Sex, Age, and Occupation	Previous Health.	Commencement of Attack.	Date of Admission.	Amount of Lung involved.	Pulse	Respiration. Temp.	TREATMENT.	Date of Convalescence. T. Period from treatment. A. Period from attack.	Date of Discharge. Number of days in hospital.	Result. Complications. Remarks.
1	L. J.	M., 47, Sailor.	Good. Stout, muscular.	Mar. 4, 1860.	Mar. 14, 1860.	⅔ Left.	92	Dyspnœa.	Leeches before admission. Small doses antimony. Blister, 16th.	March 18, end 4th day T. and 14th A.	March 22. 8 days.	Recovered.
2	W. E.	M., 29, Sailor.	Good. Stout, muscular.	Mar. 23 ,,	Mar. 29 ,,	¾ Left. Base right.	110	40 Much dyspnœa.	One gr. antimony with saline every three hours, to April 2nd, three times a day to April 3rd. Cupping, 12 oz. on 29th March.	April 3rd, end 5th day T. and 11th A.	April 7. 9 days.	Recovered.
3	E. W.	M., 19, Sailor.	Good.	April 21 ,,	April 23 ,,	¾ Right. Pleurisy.	106	40 Dyspnœa.	April 24th, 12 leeches. 25th to 28th, ⅛ gr. antimony with saline every four hours; 26th, 8 leeches; 28th, saline alone; May 2nd, 4 oz. wine; 5th, 6 oz. Two blisters. Iodine to chest, and iod. pot. internally.	May 17, end 24th day T. and 26th A.	May 25. 32 days.	Recovered. Pleurisy delayed convalescence.
4	C. H.	M., 32, Fireman.	Good.	June 1 ,,	June 4 ,,	⅔ Left.	88	Dyspnœa.	Saline every four hours; 5th, saline with ⅛ gr. antimony every four hours; 8th, every eight hours, and 4 oz. wine; 8 leeches, 5th; blister, 8th.	June 9th, end 5th day T. and 8th A.	June 26. 22 days.	Recovered.
5	J. W.	M., 26, Labourer.	Good.	June 5 ,,	June 8 ,,	½ Left.	92	36	¾ gr. antimony with saline every four hours. Blister.	June 11, end 3rd day T. and 6th A.	June 19. 11 days.	Recovered.
6	M. L.	M., 35, Porter.	Not good. Intemperate.	July 7 ,,	July 17 ,,	⅔ Left. Pleuritic effusion.	100	32	Saline with ⅛ gr. antimony every four hours; 4 oz. brandy, afterwards 2 oz. Blister.	July 21, end 4th day T. and 14th A.	August 9. 23 days.	Recovered.
7	W. T.	M., 36, Sailor.		Jan. 28, 1861.	Jan. 30, 1861.	Right. Pleurisy.	114		Antimony ¼ gr. every four hours; 31st, ⅛ gr.; Feb. 4th, carb. ammonia, 6 oz. wine; 5th, 12 oz. brandy, cupped to 8 oz.; 6th, 4 oz. brandy. Improved under stimulants. Pulse 80; respiration 20. Relapse.		Feb. 25.	Died. Extensive pleuritic effusion.

8	G. J.	M., 26, Sailor.	Not good.	Jan. 27, 1861.	Feb. 13, 1861.	½ Right. Pleuritic effusion.		Dyspnœa.	12 leeches. Blister. Saline.	Feb. 21, end 8th day T. and 25th A.	Feb. 26. 13 days.	Recovered.
9	H. H.	M., 17, Sailor.	Good.	Apr. 21 „	Apr. 24 „	Upper lobe. right.	116		April 25th to May 1st, small doses antimony.	April 28, end 4th day T. and 7th A.	May 9. 15 days.	Recovered.
10	W. K.	M., 24, Labourer.	Ailing for some weeks		May 6 „	½ Left.	100	24	Blister. 6 oz. wine. 2 grs. blue pill twice a day.	May 10, end 4th day T.	May 13. 7 days.	Recovered.
11	T. D.	M., 28, Porter.	Good. Strong, muscular.	July 10 „	July 12 „	¾ Left.	120	40	13th, cupped to 8 oz. ¼ gr. antimony every three hours; 14th, every four hours; 16th, three times a day to 18th. 17th, blister. 27th, quinine.	July 23, end 11th day T. and 13th A.	August 3. 22 days.	Recovered.
12	L. S.	M., 24. Sailor.		Dec. 9 „	Dec. 17 „	Upper lobe right.	96	40	Small doses antimony for three days. 6 oz. wine.	Dec. 28, end 11th day T. and 19th A.	Jan. 2, 1862. 15 days.	Recovered. Pneumonia of apex (tubercular?)
13	T. R.	M., 21, Hawker.	Intemperate.	Dec. 25 „	Dec. 28 „	¾ Left. Small part right.	116	60	Six leeches before admission. Small doses antimony for four days; laudanum for delirium; brandy and wine. Blister.	Jan. 5, end 8th day T. and 11th A.	Jan. 21. 24 days.	Recovered. Delirium.
14	M. McC.	F., 25.	Weak.	Dec. 25 „	Jan. 1, 1862.	½ Right. ½ Left. Bronchitis.	116	60	2nd to 6th, ⅛ gr. antimony every four hours, improved. Antimony omitted, relapse. Repeated, 7th to 11th. Stimulants.	Jan. 11, end 10th day T. and 17th A.	Feb. 13. Well Jan. 23. Kept in for weakness. 44 days.	Recovered. Bronchitis and emphysema.
15	C. W.	M., 26, Sailor.	Good. Strong, muscular.	Apr. 24, 1862.	Apr. 26 „	⅔ Left.	108		26th, ¼ gr. antimony every three hours; 27th, every four hours; omitted, 28th; blister.	May 1, end 5th day T. and 7th A.	May 9. 13 days.	Recovered.
16	G. C.	M., 25, Sailor.	Good.	Jan. 25, 1863.	Jan. 29, 1863.	⅔ Left.	109		30th to Feb. 1st, ⅛ gr. antimony every three hours; 1st to 3rd, three times a day; blister.	Feb. 3, end 5th day T. and 9th A.	Feb. 12. 14 days.	Recovered.
17	E. H.	F., 40.	Not good.	Oct. 8 „	Oct. 8 „	Right. Bronchitis.	100		Carb. ammonia and ether. 8 oz. wine; subsequently 3 oz. brandy. Cinchona.	Oct. 24, end 16th day T. and A.	Nov. 20. 43 days.	Recovered. Bronchitis. Immersion. Suicidal.

No	Name.	Sex, Age, and Occupation	Previous Health.	Commencement of Attack.	Date of Admission.	Amount of Lung involved.	Pulse	Respiration. Temp.	TREATMENT.	Date of Convalescence. T. Period from treatment. A. Period from attack.	Date of Discharge. Number of days in hospital.	Result. Complications. Remarks.
18	L. A.	M., 29, Sailor.		Nov. 22, 1863.	Nov. 22, 1863.	⅔ Left. Base right.	132 strong.	Great dyspnœa.	Admitted on 20th Nov. with bronchitis. Stimulants ordered. On 22nd, pneumonia. Stimulants continued to 24th. Aggravation of symptoms. 24th, ¼ gr. antimony with saline every four hours. Improvement.	Nov. 28, end 6th day T. and A.	Dec. 11. 19 days.	Recovered.
19	W. S.	M., 30, Labourer.	Good.	Mar. 21, 1864.	Mar. 23, 1864.	¾ Right.	120	40	23rd to 25th, ¼ gr. antimony every four hours; then ammonia, ipec. and wine. Blister.	March 27, end 4th day T. and 6th A.	April 1. 9 days.	Recovered.
20	P. F.	M., 36, Carter.	Intemperate.	Aug. 8, „	Aug. 8 „	½ Left.	120	32	9th, a grain of opium three times a day, $\frac{1}{10}$ gr. antimony every four hours; 10th opium stopped; ipec., laudanum and antimony to 13th; then wine (6 oz.) and carb. ammonia; 17th, quinine.	Aug. 16, end 8th day T. and A.	Sept. 7. 30 days.	Recovered. Gained strength slowly.
21	G. A.	M., 32, Shipwright		Jan. 11, 1865.	Jan. 11, 1865. (*See last column.*)	⅓ Left. Acute rheumatism.	92		Bicarb potash. Opium at night; 19th cinchona and potash; 27th, quinine.	Jan. 27, end 16th day T. and A.	Feb. 10. 30 days.	Recovered. Admitted with rheum. Jan. 8. Pneum. 11th.
22	D. M.	M., 55, Fireman.	Good.	Jan. 21 „	Jan. 23 „	½ Left. ¼ Right.	120	40	23rd, brandy and ammonia; worse at night. Small doses antimony with saline and ipec. Improvement, 25th, antimony stopped; an oz. of wine every three hours; worse, 27th antimony resumed, improved; 31st, antimony omitted; 6 oz. wine.	Feb. 1, end 9th day T. and 11th A.	Feb. 22. 30 days.	Recovered. Stimulants aggravated symptoms. Antimony gave relief.
23	J. D.	M., 18, Sailor.	Good.	Feb. 19 „	Feb. 24 „	¾ Left.	120	76	24th to March 4th, 6 oz. brandy; 5th, 5 oz., and 6th, 6 oz. wine; Feb. 24th to Mar. 7th, carb. ammonia and ipec. Quinine.	March 4, end 8th day T. and 13th A.	March 17. 21 days.	Recovered.
24	J. M'N.	M., 20, Labourer.	Good.	Nov. 13 „	Nov. 17 „	½ Right.	116	44	Ipec., morphia and 8 oz. wine to 25th; then quinine and 6 oz. wine; blister.	Nov. 25, end 8th day T. and 12th A.	Jan. 5, 1866. 49 days.	Recovered. Gained strength slowly.

25	C. A.	M., 40, Sailor.		Nov. 14, 1865.	Nov. 18, 1865.	¾ Right.	120	48	18th, ½ gr. antimony every three hours; 19th, omitted, 6 oz. wine; 20th, 8 oz.; 21st, 12 oz.; 29th, 10 oz.; Dec. 9th, 6 oz.; Dec. 2nd, quinine. Two blisters.	Nov. 25, end 7th day T. and 11th A.	Dec. 16. 28 days.	Recovered.
26	B. M.	M., 27, Labourer.	Good.	Jan. 5, 1866.	Jan. 9, 1866.	Upper part right.	120		Carb. ammonia, ipec. and spirits chlorof.; 6 oz. wine; 22nd, quinine. Blister.	Jan. 14, end 5th day T. and 9th A.	Jan. 30. 21 days.	Recovered.
27	T. C.	M., 23, Labourer.		Ailing for some time.	Jan. 25 ,,	Right. Pleurisy effusion.	108		Carb. ammonia, sp. chlorof., and squills Two blisters. Feb. 3rd, quinine and wine.	Feb. 3, end 9th day T.	Feb. 28. 34 days.	Recovered.
28	M. J.	M., 28, Shoemaker		Jan. 29, 1866.	Feb. 1 ,,	½ Right. ½ Left. Pleuritic effusion.	140	Great dyspnœa.	1st to 8th, carb. ammonia and squills; 6 oz. brandy to 6th, then 4 oz.; 8th, quinine. Blister.	Feb. 8, end 7th day T. and 11th A.	March 3. 30 days.	Recovered. Very severe case. Improved rapidly under stimulants.
29	A. M.	M., 30, Sailor.			Feb. 2 ,,	⅓ Left.	112		Carb. ammonia and sp. chlorof. to 10th, then quinine; 6 oz. brandy 2nd to 15th, then 6 oz. wine.	Feb. 10, end 8th day T.	Feb. 20. 18 days.	Recovered.
30	J. R.	M., 27, Labourer.	Not good.		Mar. 2, 1866.	½ Left. Gangrene.	120		Carb. ammonia and sp. chlorof.; 6 oz. brandy, afterwards 8 oz.; wine, quinine and iron.	Mar. 26, end 24th day T.	May 17. 52 days.	Recovered. Gangrene of lung, profuse, very fœtid expectoration. Great prostration.
31	P. H.	M., 31, Groom.		Feb. 26, 1866.	Mar. 3 ,,	½ Left. Base right. Pleurisy.	116		3rd to 10th, carb. ammonia, ipec. and sp. chlorof., then quinine; 4th to 12th, 6 oz. wine.	March 9, end 6th day T. and 11th A.	March 12. 9 days.	Recovered. Systolic murmur at base of heart.
32	T. F.	M., 24, Sailor.	Good.	Mar. 16 ,,	Mar. 24 ,,	½ Right.	86		Carb. ammonia and sp. of chloroform. Blister.	March 31, end 7th day T. and 15th A.	April 1. 8 days.	Recovered.
33	H. A.	M., 19, Carter.		April 26 ,,	May 3 ,,	½ Right.	120		Tincture of iron, sp. chlor. and quinine; 6 oz. wine. Blister.	May 8, end 5th day T. and 12th A.	June 9. 37 days.	Recovered.

No	Name.	Sex, Age, and Occupation	Previous Health.	Commencement of Attack.	Date of Admission.	Amount of Lung involved.	Pulse	Respiration. Temp.	TREATMENT.	Date of Convalescence. T. Period from treatment. A. Period from attack.	Date of Discharge. Number of days in hospital.	Result. Complications. Remarks.
34	R. H.	M., 26, Ship Carpenter.	Not good.	May 25, 1866.	May 26, 1866.	Whole of right. Upper part most.	140	50	27th, carb. ammonia and sp. chlorof.; 29th, ammonia, ipec. and saline; 8 oz. wine; 30th, 12 oz. wine; 31st, 8 oz. brandy. June 1st, 8 oz. wine.	June 4, end 9th day T. and 10th A.	August 2. 69 days.	Recovered. Delirium. Hæmoptysis. Tuberculosis? Kept in on account of weak state and phthisical symptoms
35	M.O'H.	M., 31, Labourer.		Ailing for some time.	Aug. 6 ,,	⅔ Left.	100		Carb. ammonia, ipec.; 8 oz. wine; subsequently quinine.	Aug. 14, end 8th day T.	August 14. 8 days.	Recovered.
36	T. P.	M., 14, Sailor.	Good.		Oct. 2 ,,	Upper part. left.	120		2nd to 6th, ipec. and squills, no improvement, pulse kept up; 6th, carb. ammonia and 4 oz. wine, improvement; 18th, quinine; 20th, 6 oz. wine; 23rd, iron and cod-liver oil.	Oct. 18, end 16th day T.	Nov. 12. 40 days.	Recovered. Kept in on account of phthisical symptoms Went out well.
37	R. R.	M., 23, Sailor.	Intemperate.	Oct. 15, 1866.	Oct. 18 ,,	½ Right.	120		19th to 27th, carb. ammonia and spirits chlorof. with saline, 8 oz. brandy; 27th to Nov. 1st, 6 oz. brandy; 25th, quinine.	Oct. 24, end 6th day T. and 9th A.	Nov. 9. 21 days.	Recovered.
38	R. R.	M., 43, Porter.	Good.	Jan. 28, 1867.	Feb. 1, 1867.	¾ Left.	110	38	1st, carb. ammonia, spirits of chlor. and 4 oz. brandy; 2nd, 6 oz. brandy; 3rd, 8 oz., continued to 9th; then 6 oz.; 12th, quinine and wine.	Feb. 8, end 7th day T. and 10th A.	Feb. 26. 25 days.	Recovered, Delirium
39	P. C.	M., 20, Militia Man.	Good.	April 25 ,,	April 30 ,,	¾ Left. Base right.	110	52	⅛ gr. antimony with saline for two days; worse. May 2nd, carb. ammonia, ipec. and 6 oz. wine; improved.	May 6, end 6th day T. and 11th A.	May 16. 16 days.	Recovered.
40	W. T.	M., 6.		May 10 ,,	May 13 ,,	⅔ Right.	120		Ammonia, ipec. and 1 oz. brandy.	May 17, end 4th day T. and 8th A.	May 21. 8 days.	Recovered.
41	T. K.	M., 16, Grocer.	Not good. Hæmoptysis.	Ill ten days with acute rheumatism.	June 13 ,,	¾ Left.	132	40	14th, 8 oz. wine; 15th, 12 oz.; 16th, 16 oz.; 18th, brandy 12 oz., to 26th; then 10 oz.; 28th, 8 oz.; 29th, 10 oz.	June 27, end 14th day T.	August 5. 53 days.	Recovered. Rheumatic fever and en-

									wine. During acute stage no medicine except opium. 29th, quinine, and subsequently iron.			docarditis. Extreme exhaustion.
42	A. T.	M., 24, Sailor.			Aug. 28, 1867.	¾ Right. Small part left.	96		29th, 4 oz. brandy; 30th, 8 oz. wine; 31st, 12 oz. Carb. ammonia and cinchona.	Sept. 2, end 5th day T.	Sept. 17. 20 days.	Recovered.
43	J. M.	M., 28, Sailor.		Ill some time.	Sept. 18 ,,	½ Right.	114		19th, Carb. ammonia and cinchona; 26th, 6 oz. wine; 28th, iodide of pot. wine continued.	Oct. 1, end 11th day T.	Oct. 12. 24 days.	Recovered. Neglected case.
44	T. F.	M., 44, Fireman.	Good.	Nov. 29, 1867.	Dec. 1 ,,	½ Right. Pleurisy. Effusion.	120	60	Carb. ammonia and ipec., one gr. opium thrice a day; 4th, 4 oz. brandy, opium at night only; 7th, ammonia and ether.	Dec. 7, end 7th day T. and 9th A.	Dec. 12. 13 days.	Recovered.
45	S. G.	M., 18, Labourer.	Good.	Aug. 10, 1868.	Aug. 14, 1868.	Nearly whole left. Pleuritic effusion.	124	50	14th, Carb. ammonia and cinchona, 6 oz. brandy: 15th, 12 oz. brandy; 17th, 8 oz.; 24th, 6 oz.; 28th, 5 oz.; omitted 30th; opium for diarrhœa.	Aug. 23, end 9th day T. and 13th A.	Sept. 25. 32 days.	Recovered. Strumous aspect. Severe diarrhœa.
46	H. B.	M., 28, Merchant.	Not good. Hæmoptysis.	Oct. 1, ,,	Oct. 1 ,, (*See last column.*)	¾ Left.	128		Carb. ammonia and cinchona; 6 oz. wine.	Oct. 14th, end 13th day T. and A.	Oct. 23. 23 days.	Recovered. Admitted Sept. 18th, with severe hæmoptysis; ceased 27th. Pneumonia, Oct. 1st.
47	P. D.	M., 32, Fireman.			Sep. 14, 1868.	⅓ Right. Bronchitis.	100		Carb. ammonia, ipec., and tinct. cinch.; 8 oz. wine.	Sept. 22, end 8th day T.	Oct. 13. 29 days.	Recovered.
48	J. M.	M., 32, Sailor.	Good.	Feb. 18, 1869.	Feb. 20, 1869.	⅔ Left.	92		20th, Carb. ammonia, cinch. and squills; 23rd, ammonia and sp. chlor., 6 oz. wine.	Feb. 25, end 5th day T. and 7th A.	March 13. 21 days.	Recovered
49	J. R.	M., 26, Porter.	Not good.	Mar. 29 ,,	April 3 ,,	½ Left. Pleurisy.	120		Carb. ammonia, cinch., 4 oz brandy; 7th, iod. pot., cinch.; blister; 10th, quinine; 4 oz. wine.	April 9, end 6th day T. and 11th A.	April 16. 13 days.	Recovered. History of cough and hæmoptysis.
50	J. R.	M., 45, Sailor.		Apl. 28 ,,	May 1 ,,	Upper lobe right. Pleurisy.	120		4 oz. of brandy, carb. ammonia and sp. chlor.; 6th, tinct. cinch.; 13th, quina and iron, 6 oz. wine.	May 10, end 10th day T. and 13th A.	May 28. 28 days.	Recovered. Valvular disease of heart.

No.	Name.	Sex, Age, and Occupation	Previous Health	Commencement of Attack.	Date of Admission.	Amount of Lung involved.	Pulse	Respiration. Temp.	TREATMENT.	Date of Convalescence. T. Period from treatment. A. Period from attack.	Date of Discharge. Number of days in hospital.	Result. Complications. Remarks.
51	R. W.	M., 32, Sailor.	Not good.		June 8, 1869.	⅓ Left.	90	32	Carb. ammonia and cinch.; 10th, 6 oz. wine; 11th, 8 oz. wine, ammonia and sp. chlor.; 15th, 4 oz. wine; blister; July 1st, iod. pot., cinch.	June 13, end 5th day T.	July 15. 37 days.	Recovered. Had rheumatism.
52	J. McC.	M., 31, Carter.	Not good.	June 4, 1869.	June 10 ,,	Base left. Bronchitis.			Carb. ammonia and sp. chlor.; 18th, iron and quina; 6 oz. brandy, 10th to 16th; then 4 oz.	June 15, end 5th day T. and 11th A.	June 23. 13 days.	Recovered.
53	C. A.	M., 45, Cook.		July 11 ,,	July 11, ,,	Whole left	150	Great dyspnœa. 56	Carb. ammonia and sp. chlor.; 11th, 6 oz. brandy; 12th, 8 oz.; 18th, 6 oz.; 21st, 8 oz. wine.	July 17, end 6th day T. and 6th A.	August 6. 26 days.	Recovered.
54	E. B.	M., 36, Labourer.	Was recovering from Measles.	Measles 11 days before admission.	Oct. 27, ,,	Base left. Pleurisy.	112		Carb. ammonia, sp. chlor. and cinchona; 6 oz. wine. Quinine. Blister.	Oct. 31, end 3rd day T.	Dec. 1st. 35 days.	Recovered.
55	H. E.	M., 24, Boiler Maker.	Good.	Dec. 16, 1869.	Dec. 21, ,,	Whole of right. Bronchitis.	126	48	Carb. ammonia and sp. chlor.; 8 oz. brandy; 22nd, 9 oz.		Dec. 23.	Died; admitted in a sinking state. P.M.
56	O. A.	M., 22, Sailor.	Not good. Suffering from Dysentery.	Dec. 31 ,,	Treatment began Jan. 3, 1870.	Half right.	112		Carb. ammonia and sp. chlor.; 8 oz. wine; 15th, quinine.	Jan. 9, end 6th day T. and 9th A.	March 22. 72 days.	Recovered. Remained in hospital in consequence of Dysentery.
57	L. R.	M., 40, Sailor.			Jan. 5, ,,	⅓ Left. Pleurisy.	116		Carb. ammonia and sp. chlor.; 6 oz. wine. Blisters.	Jan. 18, end 13th day T.	Several weeks.	Recovered. Wasting Palsy; remained in hospital a long time on that account.
58	J. T.	M., 36, Sailor.	Good.		Mar. 2, ,,	Small part left.			Carb. ammonia and sp. chlor. Blister.	March 10, end 8th day T.	March 29. 27 days.	Recovered. Mild attack, followed injury to side.

59	M. C.	M., 45, Sailor.	Good.	Apr. 23, 1870.	Apr. 26, 1870.	Base left.	106		Carb. ammonia and sp. chlor.; 4 oz. wine. Blister.	May 1, end 5th day T. and 8th A.	May 24. 28 days.	Recovered.
60	N. C.	M., 8.		July 4 ,,	July 7 ,,	Base right. Pleurisy.	132		Carb. ammonia and saline; 1½ oz. brandy. Iodide potas. and tonics.	July 16, end 9th day T. and 12th A.	August 8. 32 days.	Recovered.
61	J. F.	M., 32, Traveller.	Good.		Jan. 19, 1871.	Base right. Bronchitis.	120		Carb. ammonia and sp. chlor.; 6 oz. wine. Blister.	Feb. 11, end 23rd day T.	Feb. 27. 39 days.	Recovered. Attack followed immersion.
62	J. M'D.	M., 32, Bookbinder		Treatment began Oct. 1, 1871.	Sept. 22, ,, Acute rheumatism.	Base right.	104		3rd Oct., alkalies, carb. ammonia, and cinchona; 6th, 4 oz. brandy; 13th, 8 oz. wine.	Oct. 13, end 13th day T.	Nov. 7. 46 days.	Recovered. Kept in for rheumatism.
63	C. W.	F., 20, Servant.		Treatment began Dec. 5, 1871.	Nov. 28, ,, Acute rheumatism.	½ Left. Peri- and endocarditis.	130	30	Alkalies at first. Dec. 1st, 4 oz. brandy; 6th, carb. ammonia and 6 oz. brandy.	Dec. 18, end 13th day T.	Jan. 23. 57 days.	Recovered. Acute rheumatism, peri- and endocarditis.
64	S. G.	M., 15, Seaman.		Treatment began Dec. 27, 1871.	Dec. 24, ,,	½ Right. Acute rheumatism; endocarditis.	108	40	27th, opium; 3 oz. brandy; 30th, 6 oz.; carb. ammonia and potash; Jan. 3rd, 3 oz. brandy.	Jan. 10, 1872, end 14th day T.	March 13. 79 days.	Recovered. Kept in for endocarditis.
65	W. S.	M., 33, Shipwright.	Good.	Dec. 23 ,,	Dec. 30, ,,	Whole right.	132	32 101·5°	Carb. ammonia and sp. chlor.; 6 oz. brandy. Cinchona.	Jan. 6, end 7th day T. and 14th A.	Feb. 23. 55 days.	Recovered. Had two attacks of erysipelas of face.
66	C. K.	F., 22.	Good.	Jan. 3, 1872.	Jan. 10, 1872.	⅓ Right.	124		Carb. ammonia; sp. chlor; 4 oz. brandy.	Jan. 15, end 5th day T. and 12th A.	Jan. 19. 9 days.	Recovered.
67	R. T.	M., 17, Ship Steward.	Not good.	Jan. 13 ,,	Jan. 17, ,,	½ Left.	136	35 106°	Carb. ammonia; sp. chlor; 6 oz. brandy.	Jan. 24, end 7th day T. and 11th A.	Feb. 22. 36 days.	Recovered.
68	E. G.	F., 15.			Jan. 20, ,,	⅓ Left. Rheumatic carditis.	120	50 Temp. 103·5°	Carb. ammonia; 1½ oz. brandy.	Feb. 7, end 16th day T.	March 8. 48 days.	Recovered. Rheumatic fever, Peri- and endocarditis.

No.	Name.	Sex, Age, and Occupation	Previous Health.	Commencement of Attack.	Date of Admission.	Amount of Lung involved.	Pulse	Respiration. Temp.	TREATMENT.	Date of Convalescence. T. Period from treatment. A. Period from attack.	Date of Discharge. Number of days in hospital.	Result. Complications. Remarks.
69	E. Y.	F., 20.			Jan. 20, 1872.	Base right.			Carb. ammonia; cinchona; 4 oz. wine.	Jan. 30, end 10th day T.	Feb. 23. 32 days.	Recovered.
70	A. O'R.	M., 22, Tailor.		Jan. 27, 1872.	Feb. 3, ,,	½ Left.	90		Carb. ammonia; sp. chlor; 4 oz. brandy.	Feb. 6, end 3rd day T. and 10th A.	Feb. 19. 16 days.	Recovered.
71	E. D.	F., 28.		Feb. 2 ,,	Feb. 3 ,,	½ Left.			Carb. ammonia and sp. chlor; 3 oz. brandy.	Feb. 8, end 5th day T. and 6th A.	Feb. 23. 20 days.	Recovered.
72	P. R.	F., 29.	Good.	Feb. 24 ,,	Feb. 29 ,,	Base right. Bronchitis.	110		Carb. ammonia and cinchona; March 3rd, 6 oz. wine.	Mar. 10, end 10th day T. and 15th A.	April 5. 36 days.	Recovered.
73	J. O.	M., 42. Labourer.	Good.	Apr. 13 ,,	Apr. 20 ,,	¾ Right.	110		Carb. ammonia; 6 oz. brandy; 26th, 4 oz.	April 27, end 7th day T. and 14th A.	May 23. 33 days.	Recovered.
74	T. A.	M., 26, Brewer.			Sept. 21 ,,	Base right.			Carb. ammonia; 6 oz. wine.		Oct. 28. 37 days.	Recovered.
75	G. P.	F., 27, Servant.	Not good.		Sept. 28 ,,	Base right.	116		Carb. ammonia and cinchona; 6 oz. wine.		Oct. 24. 26 days.	Recovered.
76	S. B.	F., 22.		Treatment began Oct. 22, 1872.	Oct. 12 ,, Rheumatic fever. Pericarditis.	Base. both.	104		Carb. ammonia and carb. potash; cinchona.	Oct. 30, end 8th day T.	Nov. 13. 32 days.	Recovered.
77	J. W.	M., 28, Steward.	Good.	Apr. 22, 1873.	Admitted Apr. 12, 1873, with congestion of liver.	½ Right. Pleurisy.	104		Carb. ammonia and sp. chlor; April 24th, 6 oz. brandy; 26th, 4 oz.; iod. of pot: May 2nd, brandy omitted.	April 28, end 6th day T. and 6th A.	May 3. 11 days from commencement of pneumonia.	Recovered.

78	J. B.	M., 19, Striker.	Good.	May 9, 1873.	May 15, 1873.	¾ Right.	100	60 101°	Carb. ammonia and cinchona, 6 oz. brandy; 19th, 5 oz. brandy; 20th, 4 oz.	May 18, end 3rd day T. and 9th A.	May 26. 9 days.	Recovered.
79	J. W.	M., 20, Labourer.	Good.	May 11 ,,	May 15 ,,	½ Left.	90	101°	Carb. ammonia and sp. chlor.	May 18, end 3rd day T. and 7th A.	May 28. 13 days.	Recovered.
80	G. M.	M., 24, Seaman.	Good.	Feb. 9, 1874.	Feb. 11, 1874.	⅜ Left. Base right.	128	56 104°	Carb. ammonia, 6 oz. brandy till 22nd, then gradually diminished.	Feb. 21, end 10th day T. and 12th A.		Recovered. Valv. disease of heart.
81	J. H.	M., 63, Gentleman	Bad.		June 27 ,,	Whole of right. Base left.	100	36 102°	Carb. ammonia, cinchona, and 6 oz. brandy.		July 6.	Died. Phthisis. Cavity at apex.
82	H. L.	F., 47, Cook.	Not good. Bronchitis.	Nov. 30 ,,	Dec. 4 ,,	Whole of right.	134		Carb. ammonia, ipecac, ether, and 6 oz. brandy.		Dec. 11.	Died. Diarrhœa.
83	C. W.	M., 34, Seaman.		Dec. 17 ,,	Dec. 23 ,,				Carb. ammonia, sp. chlor., 3 oz. brandy.		Jan. 28. 36 days.	Recovered.
84	H. T.	M., 44, Carter.	Not good. Asthmatic.	Dec. 25 ,,	Dec. 30 ,,	¾ Left.	116	104°	Carb. ammonia, cinch., 6 oz. brandy.		Jan. 14.	Died. Gangrene.
85	E. W.	F., 33.			Jan. 20, 1875.	Base left.	100	64 100°	Bromide pot., carb. ammonia, cinchona, 6 oz. brandy; 30th, 4 oz.	Feb. 1, end 12th day T.	Feb. 23. 34 days.	Recovered. Delirium tremens.
86	M. D.	F., 26.	Good.	3 weeks before admission. (?)	April 7 ,,	Base right. Pleurisy.	132	40 105·4°	Carb. ammonia, iod. pot., 4 oz. brandy.	April 29, end 22nd day T.	May 13. 36 days.	Recovered. Pleurisy.
87	T. W.	M., 50, Mason.	Good.		May 1 ,,	¾ Right.	132	60 104°	Carb. ammonia, 8 oz. brandy; gradually diminished.	May 5, end 4th day T.	May 26. 25 days.	Recovered.
88	E. T.	M., 24, Labourer.	Good.	Jan. 8, 1875.	Jan. 13 ,,	Base left.		102°	Carb. ammonia, 4 oz. brandy.	Jan. 18, end 5th day T. and 10th A.	Jan. 29. 16 days.	Recovered.
89	E. S.	F., 48.	Good.	June 19 ,,	June 26 ,,	¾ Left.	126	44 102·5°	Carb. ammonia, cascarilla, 4 oz. brandy; 28th, 6 oz.; 30th, 2 oz. wine; July 7th, 3 oz. brandy.	July 6, end 10th day T. and 17th A.	July 23. 27 days.	Recovered.

No.	Name.	Sex, Age, and Occupation	Previous Health.	Commencement of Attack.	Date of Admission.	Amount of Lung involved.	Pulse	Respiration. Temp.	TREATMENT.	Date of Convalescence. T. Period from treatment. A. Period from attack.	Date of Discharge. Number of days in hospital.	Result. Complications. Remarks.
90	T. B.	M., 25, Carter.	Not good.	Aug. 21, 1875.	Aug. 27, 1875.	Base left.	110	34 103·4°	Carb. ammonia, ether, 4 oz. wine.	Sept. 1, end 5th day T. and 11th A.	Sept. 4. 8 days.	Recovered.
91	W. E.	M., 22, Gardener.		Nov. 30 „	Dec. 4 „	Base left. Bronchitis.	108	36 103·6°	Carb. ammonia, cascarilla, 4 oz. brandy, cinchona.	Dec. 8, end 4th day T. and 8th A.	Dec. 18. 14 days.	Recovered.
92	E. F.	F., 50.	Good.	Three weeks before admission. (?)	Nov. 22, 1876.	Base left.	100	28 100°	Carb. ammonia, 6 oz. wine, quinine.	Dec. 1, end 9th day T.	Dec. 22. 30 days.	Recovered. Neglected case.
93	P. S.	M., 47.		Jan. 22, 1877.	Jan. 20, 1877. (*See last column.*)	Base right. Bronchitis.	132	70 104°	Carb. ammonia, ether, bromide pot., 3 oz. brandy; 31st, 6 oz. claret.	Feb. 10, end 19th day T. and 19th A.	Feb. 21. 32 days.	Recovered. Admitted for delirium tremens.
94	W. M.	M., 33.		Feb. 8 „	Feb. 14 „	Base right.			Carb. ammonia, sp. chlor., cascar., 3 oz brandy, cinchona.		March 5. 19 days.	Recovered.
95	J. C.	M., 33, Labourer.	Good.		Mar. 28 „	⅓ Left. Base right.	100	102·6°	Carb. ammonia and cinchona; 6 oz. brandy, quinine.	April 1, end 4th day T.	April 18. 21 days.	Recovered.
96	J. N.	M., 52, Seaman.			April 4 „	Right.			Carb. ammonia, cascarilla, 3 oz. brandy, quinine.	April 9, end 5th day T.	May 9. 35 days.	Recovered.
97	G. M.	M., 48, Seaman.		April 21 „	April 28 „	⅔ Right.			Carb. ammonia, cascarilla, 4 oz. brandy.	May 6, end 8th day T. and 15th A.	May 15. 17 days.	Recovered.
98	J. S.	M., 24, Labourer.	Good.	May 9 „	May 16 „	Whole of right.	126	40 103·5°	Carb. ammonia, cinchona, 6 oz. brandy. 20th, 4 oz.	May 20, end 4th day T. and 11th A.		Recovered.
99	W. L.	M., 58.			Nov. 21 „	⅓ Left.			Carb. ammonia, cascarilla.		Dec. 26. 35 days.	Recovered. Mild case; neglected. Pleurisy.

100	J. R.	M., 36.	Good.	Nov. 25, 1877.	Dec. 1, 1877.	⅔ Left. Pleuritic effusion.	108	44 103°	Carb. ammonia, sp. chlor., cascar.; cinchona, 4 oz. brandy, quinine.	Dec. 7, end 6th day T. and 12th A.	Jan. 14. 44 days.	Recovered.
101	T. S.	M., 38.		Nov. 25 ,,	Dec. 1 ,,	½ Left.	140	48 104°	Carb. ammonia, bromide pot., chloral; 6 oz. brandy; 5th, 3 oz. and pint porter.	Dec. 10, end 9th day T. and 15th A.	Dec. 29. 28 days.	Recovered. Delirium tremens.
102	E. B.	F., 37.	Good.	Dec. 9 ,,	Dec. 12 ,,	Whole of left.	140	52 104°	Carb. ammonia, sp. chlor., cascar., 4 oz. brandy; 15th, 6 oz.; 17th, quinine gr. 16 daily; iron.	Dec. 28, end 16th day T. and 19th A.	April 18. 4 months, 6 days.	Recovered. Very severe scrofulous pneumonia.
103	T. B.	M., 40.		Mar. 26, 1878.	Mar. 30, 1878.	Base left.	140	103·8°	Carb. ammonia, sp. chlor., cascarilla; brandy.		June 23. 84 days.	Recovered.
104	J. W.	M., 14.	Good.	April 7 ,,	April 10 ,,	⅓ Left.	126	44 105·5°	Carb. ammonia, acet. ammonia, sp. chlor., 2 oz. brandy.	April 14, end 4th day T. and 7th A.	April 23. 13 days.	Recovered.
105	C. O.	F., 17.	Good.	April 8 ,,	April 10 ,,	Base left.	136	36 105·4°	Carb. ammonia, sp. chlor., cascarilla; brandy.	April 15, end 5th day T. and 7th A.	April 22. 12 days.	Recovered.
106	J. H. M.	M., 7.	Good.	May 18 ,,	May 22 ,,	Base left.	140	103·8°	Carb. ammonia, sp. chlor., cascarilla; brandy.	May 26, end 4th day T. and 8th A.	June 23. 32 days.	Recovered.
107	J. T.	M., 21, Seaman.	Good.	May 26 ,,	May 29 ,,	½ Left.	112	103°	Carb. ammonia, 4 oz. brandy.	June 2, end 4th day T. and 7th A.		Recovered.
108	E. R.	F., 22.	Good.	Jan. 25, 1879.	Jan. 26, 1879.	Base left.		104·2°	Carb. ammonia, sp. chlor., cascarilla.	Jan. 31, end 5th day T. and 6th A.		Recovered.
109	E. J. M.	F., 20.		Mar. 2 ,,	Mar. 5 ,,	Both. Bronchitis.	152	40 104°	Carb. ammonia, brandy.		March 7.	Died.
110	J. E.	M., 36.	Good.	May 4 ,,	May 7 ,,	⅓ Left.		103°	Carb. ammonia, sp. chlor., cascarilla.	May 15, end 8th day T. and 11th A.	May 21. 14 days.	Recovered.
111	J. H.	M., 28,	Not good.	Oct. 31 ,,	Nov. 5 ,,	½ Left. Base right.	96	48	Carb. ammonia, sp. chlor., cascar., chloral; quinine and iron.	Nov. 12, end 7th day T. and 12th A.	Nov. 25 20 days.	Recovered. Delirium.

No.	Name.	Sex, Age, and Occupation	Previous Health.	Commencement of Attack.	Date of Admission.	Amount of Lung involved.	Pulse	Respiration. Temp.	TREATMENT.	Date of Convalescence. T. Period from treatment. A. Period from attack.	Date of Discharge. Number of days in hospital.	Result. Complications. Remarks.
112	M. M.	F., 35.	Good.	Nov. 26,1879.	Dec. 6, 1879.	½ Right. ½ Left. Bronchitis.	140	40 103°	Carb. ammonia, sp. chlor., senega., 8 oz. brandy; Dec. 13th, 4 oz.; 10th, quinine gr. 20 daily; 12th, gr. 10 daily.	Dec. 15, end 9th day T. and 19th A.	Jan. 3. 28 days.	Recovered. Aborted during the attack.
113	W. P.	M., 13.	Not good.	Jan. 3, 1880.	Jan. 3, 1880.		144	60 103·4°	Carb. ammonia, sp. chlor., cascarilla.	Jan. 9, end 6th day T. and 6th A.		Recovered. Mitral disease.
114	J. C.	M., 43.	Heart disease.	Several days before admission.	Jan. 10, „	¾ Left.			Carb. ammonia, cascarilla, quinine, wine.		Feb. 2. 23 days.	Recovered. Mitral disease.
115	S. D.	M., 18.	Good.	Mar. 25, 1880	Mar. 27, „	½ Left.	120	36 104°	Carb. ammonia, sp. chlor., cascarilla.	April 2, end 6th day T. and 8th A.	April 19. 23 days.	Recovered.
116	J. F.	M., 29, Labourer.			Sept. 11, „	No	de-	tails.	Carb. ammonia, cascarilla, quinine.		Sept. 25. 14 days.	Recovered.
117	J. L.	M., 19, Bricklayer.	Good.	May 1, 1881.	May 7, 1881.	½ Right.	106	42 103·2°	Carb. ammonia, sp. chlor., cascarilla, 4 oz. brandy.	May 12, end 5th day T. and 11th A.	May 23. 16 days.	Recovered.
118	E. J.	M., 25, Labourer.	Very intemperate.	May 8, „	May 11, „	⅔ Right. Base left.	142	48 102°	Carb. ammonia, quinine, 6 oz. brandy.		May 14.	Died.
119	J. L.	M., 19.	Good.	June 19, „	June 23, „	Base left.	144	60 103·4°	Carb. ammonia, sp. chlor., cascarilla, brandy.	June 26, end 3rd day T. and 7th A.	July 3. 14 days.	Recovered. 2nd attack. (*See* No. 117.)
120	E.McK.	F., 67.	Good.	Dec. 15, „	Dec. 17, „	½ Left.	120	30 103·2°	Carb. ammonia, 4 oz. brandy.	Dec. 20, end 3rd day T. and 5th A.	Jan. 6. 20 days.	Recovered.
121	W. A.	M., 39.		Several days before admission.	Dec. 21, „	⅓ Left.	84	28	Carb. ammonia, sp. chlor., cascarilla, bromide pot.		Jan. 21. 31 days.	Recovered. Delirium Tremens.

122	J. R.	M., 53.	Intemperate. Several attacks D. T.	Several days before admission.	Jan. 7, 1882.	Left.	90	27	Carb. ammonia, cascarilla, sp. chlor.		Jan. 21. 14 days.	Recovered. Defervesence before admission.
123	C. McE	F., 19.	Good.	Jan. 16, 1882.	Jan. 18, ,,	¾ Left. ⅓ Right.	150	64 103·6°	Carb. ammonia, cinch., 6 oz. brandy, 23rd, 4 oz.; 31st, 3 oz.	Jan. 22, end 4th day T. and 6th A.	Feb. 15. 28 days.	Recovered. Diarrhœa.
124	J. C.	M., 30, Labourer.	Good.	Several days before admission.	Jan. 11, ,,	½ Right.	90	30 100°	Carb. ammonia, cascarilla.	Jan. 17, end 6th day T.	Jan. 25. 14 days.	Recovered. Acute stage over when admitted
125	A. J.	F., 19, Servant.	Good.	Mar. 24, ,,	Mar. 27, ,,	Whole of right.	132	48 104°	Carb. ammonia, cinch., ether, brandy.	April 24, end 28th day T. and 31st A.	May 29. 63 days.	Recovered.
126	A. E.	F., 30.	Good.	April 23, ,,	April 26, ,,	Apex right.	132	46 100·2°	Carb. ammonia, 4 oz. brandy.	April 30, end 4th day T. and 7th A.	May 31. 35 days.	Recovered.
127	–. O'L.	M., 39.			May 3. ,,				Carb. ammonia, 4 oz. brandy.		May 26. 23 days.	Recovered.
128	R. A.	M., 28, Labourer.	Intemperate.	May 24, ,,	May 31, ,,	⅓ Right.			Carb. ammonia, 4 oz. brandy.		June 12. 12 days.	Recovered. Defervescence before admission.
129	M. W.	F., 18.		May 27, ,,	May 31, ,,	Apex left. Pleurisy.	132	52 104·2°	Carb. ammonia, sp. of nitre, small quantity of brandy.	June 7, end 7th day T. and 11th A.	July 17. 47 days.	Recovered.
130	M. P.	F., 17.			Nov. 25, ,,	Base both. Rheumatic fever. Pericarditis	148	46 103·6°	Carb. ammonia, iod. pot., 10 oz. brandy, gradually diminished.	Dec. 9. end 14th day T.		Recovered. Heart disease proved fatal later.
131	J. G.	M., 39, Marine Fitter.	Intemperate.	Feb. 14, 1883. Injury to side	Feb. 17, 1883.	Whole of right. ½ Left. Pleurisy.	126	48 101·6°	Carb. ammonia, cinch., 6 oz. brandy; 19th, 8 oz.; 20th, quinine, gr. 10 daily.		Feb. 21.	Died. Tubercles at right apex.

No.	Name.	Sex, Age, and Occupation	Previous Health.	Commencement of Attack.	Date of Admission.	Amount of Lung involved.	Pulse	Respiration. Temp.	TREATMENT.	Date of Convalescence. T. Period from treatment. A. Period from attack.	Date of Discharge. Number of days in hospital.	Result. Complications. Remarks.
132	T. J.	M., 40, Brewer's Labourer.	Intemperate.		Apr. 11, 1883.	Apex.	120	104·6°	Carb. ammonia, digitalis, chloral, bromide pot.		May 1.	Died.
133	H. R.	M., 20, Labourer.	Not good. Intemperate.	May 23, 1883.	May 26 ,,	Apex right.	124	44 102·6°	Carb. ammonia, cinch., 29th, quinine, gr. 12; June 5th, gr. 6, daily.	June 2, end 7th day T. and 10th A.	June 20. 25 days.	Recovered. Delirium.
134	W. L.	M., 22, Butcher.	Intemperate.	May 27, ,,	June 2 ,,	⅓ Right. Base left.		102·4°	Carb. ammonia, 16th, quinine.		June 20. 18 days.	Recovered.
135	R. S.	F., 48.	Not good.		June 15 ,,	⅔ Right. ¼ left.	100	44 102·6°	Carb. ammonia, cinch., quinine, 4 oz. brandy; later 6 oz.	June 26, end 11th day T.	Oct. 15. 4 months.	Recovered. Became chronic. Bronchiectasis.
136	C. S.	M., 54.		June 7, ,,	June 16 ,,	Lower lobe. right.	86	32 101°	Carb. ammonia, cinch., 3 oz. brandy; July 18th, 6 oz.; 23rd, 4 oz.	June 29, end 13th day T. and 22nd A.	July 31. 45 days.	Recovered. Acute symptoms subsided before admission.
137	T. W.	M., 28.			Aug. 1 ,,		100	48 103°	Carb. ammonia, 4 oz. brandy.	Aug. 7, end 6th day T.	Aug. 20. 19 days.	Recovered.
138	M. O'N.	F., 56, Housekeeper.		Three weeks before admission. (*See last column.*)	July 17, 1885.	Base both. Acute rheumatism.	Very quick.	108·8°	Salicyl. sod., quinine, brandy.		Aug. 7.	Died. Rheumatic fever. Hyperpyrexia.
139	M. H.	F., 22.			Jan. 9, 1884.	Base both. Acute rheumatism.	128	44 103·5°	Carb. ammonia, salicyl. sod., digitalis.	Jan. 29, end 20th day T.	April 7. 89 days.	Recovered. Old heart disease.
140	E. M.	F., 17.	Good.	Feb. 13, 1886.	Feb. 20, 1886.	Base right		101·4°	Carb. ammonia, cascarilla.	Feb. 27, end 7th day T. and 14th A.	March 9. 17 days.	Recovered.

141	J. S.	M., 44, Labourer.			Feb. 13, 1884.				No details.		Feb. 15.	Died.
142	J. C.	M., 7, Schoolboy.			Mar. 12 ,,	Right. Pleuritic effusion.			Carb. ammonia, quinine.		April 30. 49 days.	Recovered.
143	H. B.	M., 37, Shoemaker			June 25 ,,	Base right	130	26 105°	Carb. ammonia, 4 oz. brandy; 28th, 8 oz.; July 2nd, 4 oz. wine.	July 1, end 6th day T.	July 7. 12 days.	Recovered.
144	J. W.	M., 33, Labourer.			Feb. 14, 1885.	Left.	128	28 104°	Carb. ammonia, 4 oz. brandy; 17th, 2 oz.		March 14. 28 days.	Recovered.
145	A. A.	M., 35, Plumber.			Dec. 29, 1883.	Whole of right. Pleurisy.	116	28 103°	Carb. ammonia, quinine, brandy.	Jan. 1, end 3rd day T.	Jan. 28. 30 days.	Recovered. Pleurisy.
146	G. T.	M., 31, Dock Labourer.			Feb. 12, 1884.	Apex left.	126	50 104·7°	Carb. ammonia; 15th, quinine, gr. 10, daily, brandy.	Feb. 25, end 13th day T.	March 13. 30 days.	Recovered. Delirium.
147	M. B.	F., 28.	Good.		Mar. 3, 1886.	Base left.		103·2°	Carb. ammonia, cascarilla.	March 6, end 3rd day T.	March 25. 22 days.	Recovered.
148	W. C.	M., 17, Paper Ruler.	Good.	Feb. 26, 1886.	Mar. 5 ,,	¾ Right.	120	102°	Carb. ammonia, cinch., 3 oz. brandy; March 8th, quinine, 2 oz. brandy.	Mar. 10, end 5th day T. and 12th A.	March 27. 22 days.	Recovered. Delirium.
149	J. W.	M., 39, Seaman.			June 30 ,,	Apex right.	110	44 104°	Carb. ammonia, cascarilla, quinine, gr. 6, daily; stimulants.	July 7, end 7th day T.	July 26. 26 days.	Recovered.
150	E. E.	M., 29, Carter.		Ill nearly a month.	Oct. 20 ,,	Base left.	78	102°	Carb. ammonia; 27th, quinine.	Nov. 4, end 15th day T.	Nov. 15. 26 days.	Recovered.
51	T. B.	M. 6.			Nov. 24 ,,	Apex right chiefly.	144	54 103·2°	Aromat. sp. ammonia, cinchona, 1 oz. brandy.	Nov. 30, end 6th day T.	Dec. 14. 20 days.	Recovered.

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